

The Consumption of Alcohol and Neurocognitive Function in Youth

Michelle Dwyer

BA (Hons)

A report submitted in partial requirement for the degree of Master of

Psychology (Clinical) at the University of Tasmania

Word Count: 9,998

I declare that this research report is my own work and that, to the best of my knowledge and belief, it does not contain material from published sources without proper acknowledgement, nor does it contain material which has been accepted for the award of any other higher degree or graduate diploma in any university.

Signed:_____

Date: 21/12/2015

Acknowledgements

I would like to thank those who have offered ongoing support and guidance throughout the completion of this thesis. I wish to express my sincere gratitude to my supervisors Raimondo Bruno and Allison Matthews for their wisdom, advice, and encouragement through this process. Their insightful comments and helpful suggestions have been extremely valuable. I would also like to thank my fellow master's students for their kindness, moral support, warm friendships and for the stimulating discussions throughout the past two years. You all have made this master's experience a very enjoyable one and I have made some wonderful friendships that I hope will continue into the future. Thank you to my other friends and family for providing unconditional support, advice, understanding and love. I also wish to thank Janette Smith and Raimondo Bruno for providing participant contacts from the larger ARC study. And finally, thank you to all of the participants who have taken part in this study, without you this thesis would not be possible.

Table of Contents

Abstract.....	1
Introduction.....	2
Frontal Lobes and Executive Functioning.....	2
Frontal and Parietal lobes, Executive Functioning, Binge Drinking and Adolescence	4
Males and Binge Drinking.....	6
Interference Control and the Flanker/Go-nogo Task.....	7
Electrophysiology of Interference Control in Binge Drinkers.....	9
The Current Study.....	11
Method.....	14
Participants.....	14
Apparatus / Materials.....	16
Electrophysiological Recording.....	17
Procedure.....	18
Design & Data Analysis.....	19
Results.....	20
Reaction Time.....	20
Accuracy.....	20
Electrophysiological Data.....	21
Discussion.....	30
Binge Drinking and Interference Control On the Flanker/Go-nogo Task.....	32
Binge drinking and Electrophysiological Responses.....	33
Limitations and Future Research.....	36
Practical Implications.....	39

Conclusion.....	41
References.....	43
Appendix A: Email to Participants.....	54
Appendix B: Screening Questionnaire.....	57
Appendix C: Human Research Ethics Committee (HREC) Approval from the University of Tasmania.....	65
Appendix D: Information Sheet	68
Appendix E: Informed Consent.....	72
Appendix F: Data Analyses.....	77

List of Tables and Figures

Table 1. Means and Standard Deviations (%) of Alcohol Use for Low Level and Binge Drinkers.....	16
Table 2. Mean (<i>SD</i>) and 95% CI for Reaction Time and Accuracy for Low Level and Binge Drinkers.....	21
Figure 1. Grand mean averaged waveforms (left hand side) and difference waveforms (right hand side) for low level drinkers.....	23
Figure 2. Grand mean averaged waveforms (left hand side) and difference waveforms (right hand side) for binge drinkers.....	24
Figure 3. Mean peak N2 amplitude for low level and binge drinkers at frontocentral (FZ, FCZ, CZ) electrode sites. Error bars represent 95% confidence.....	25
Figure 4. Mean peak N2 amplitude for go congruent, go incongruent and nogo incongruent trials across FZ, FCZ and CZ electrode sites. Error bars represent 95% confidence.....	26
Figure 5. Mean peak P3 amplitude for go congruent, go incongruent and nogo incongruent trials across PZ, CZ and FZ electrode sites. Error bars represent 95% confidence.....	29
Table 3. Pairwise Comparisons for each Trial Type at each Electrode Site (PZ, CZ, FZ)	30

Abstract

Binge drinking appears to be associated with frontal lobe damage and executive function impairments in adults (Wilcox et al., 2014). It is suggested that as adolescent's brains have not yet reached full development (Petit et al., 2013), they may be particularly vulnerable to these effects. The current study aimed to investigate if binge drinking is associated with deficits in behavioral and psychophysiological measures of executive function, interference control. Twenty-two adolescent males (11 binge drinkers and 11 low level drinkers) were recruited. All participants completed a flanker/go-nogo task which required them to respond to target stimuli whilst withholding their responses to irrelevant stimuli. Binge drinkers did not show a significantly higher number of errors or longer reaction times on the flanker/go-nogo task in comparison to low level drinkers. Additionally, binge drinkers did not show significantly reduced N2 and P3 amplitude at frontal and parietal electrode sites in comparison to low level drinkers. However, low level drinkers showed significantly greater N2 amplitude at frontal in comparison to central electrode sites, whereas binge drinkers did not show this difference. The results from the current study suggest that binge drinkers may employ more widespread recruitment of electrophysiological resources to inhibit their responses and attend to stimuli with no increase in task performance, in comparison to low level drinkers. This study has identified that early intervention may be especially important for adolescent males in order to attempt to reduce binge drinking and protect adolescents from cognitive difficulties associated with binge drinking.

Problematic alcohol consumption can have long-term negative consequences on brain structure and function, as well as cognitive ability (Pfefferbaum, Adalsteinsson, & Sullivan, 2006). Adolescent drinkers, in particular, are at risk as their brains have not yet reached full development, and are more sensitive to the effects of alcohol induced damage (Petit, Maurage, Kornreich, Verbanck, & Campanella, 2013). Furthermore, adolescent drinkers are more likely to engage in health risk behaviours such as smoking cigarettes, engaging in unsafe sexual behaviours or driving/riding in a car with an intoxicated driver (Miller, Naimi, Brewer & Jones, 2007). The National Health and Medical Research Council (NHMRC) guidelines suggest that adolescents aged between 15 and 17 years old should delay drinking for as long as possible and individuals under the age of 16 should avoid alcohol completely, as alcohol use can be particularly damaging for this age group (NHMRC, 2009).

However, consumption of alcohol is normative for adolescents aged 18 and under in Australian society. An estimated 70% of adolescents have consumed a full serve of alcohol by the age of 15 and approximately 30% of Australians aged 15 to 17 years old have engaged in binge drinking behavior at least once (Roche et al., 2007). While there is no consensus around the minimum amount of standard drinks needed to confirm a binge drinking session, literature considers a binge drinking session as consuming approximately four or more standard alcoholic drinks in a short period of time (Crego et al., 2012; NHMRC, 2009). Research has shown that adolescents who engage in regular binge drinking have blunted development of both white and gray matter in their brain, as well as frontal lobe damage compared to controls (De Bellis et al, 2005; Luciana, Collins, Muetzel, & Lim, 2013).

Frontal Lobes and Executive Functioning

The frontal lobes are crucial in the development of an individual's personality, emotional processes and behaviours (Russo, 2003). They are generally responsible for higher order cognitive processes and executive functions. Executive functions consist of the ability to make decisions, consciously process information, control impulses, problem solve and self-regulate (James, Reichelt, Carlson & McAnaney, 2008; Sneider & Silveri, 2015). Individuals with frontal lobe damage have difficulties with executive functions such as making everyday decisions about finances and employment (Clark & Manes, 2004). Furthermore, the frontal lobes also play an important role in attentional control and working memory (James et al., 2008). Therefore, individuals with frontal lobe damage may have difficulties retaining information and performing mental operations on information presented briefly.

Heavy alcohol use and binge drinking can cause damage to the brain, particularly to the frontal lobes (Pfefferbaum, Adalsteinsson, & Sullivan, 2006). Mashhoon et al. (2014) compared the brain structure of the prefrontal cortex in binge drinking and light drinking young adults using high-resolution magnetic resonance imaging (MRI). They found that the prefrontal cortex was thinner for binge drinkers than for light drinkers. This suggests that binge drinking appears to be associated with cortical thinness and damage to particular areas of the frontal lobe. It is also suggested that individuals who engage in excessive alcohol use may experience profound executive functioning impairments due to frontal lobe damage. Scaife and Duka (2008) explored the link between executive difficulties and drinking among binge drinking and non-binge drinking young adults. It was found that binge drinkers showed impairments in working memory, visual discrimination and attentional shift in comparison to non-binge drinkers. It is suggested that binge drinking may be

particularly harmful during adolescence, as the frontal lobes of the brain are undergoing important changes and growth during this developmental period, and are therefore more sensitive to brain damage and executive dysfunction (Medina et al., 2008).

Frontal and Parietal lobes, Executive Functioning, Binge Drinking and Adolescence

The brain does not fully develop until approximately the mid-twenties (Pujol, Vendrell, Junque, Mari-Vilalta & Capdevila, 1993), in particular, the frontal and parietal lobes are not fully developed until after adolescence (Giedd et al., 1999). It is therefore suggested that, as executive functions and various parts of the brain are not fully developed, adolescents' ability to make adequate decisions taking into consideration the consequences associated with their actions, and their ability to control impulsive behaviours may be limited (Casey & Jones, 2010). As a result, this makes adolescents prone to engage in risk taking and sensation seeking behaviours (Dayan, Bernard, Olliac, Mailhes & Kerrmarec, 2010). The lack of these cognitive control processes increases the likelihood of adolescents becoming dependent on alcohol and other substances (Rubio et al., 2008). Therefore, it is suggested that adolescents are likely to engage in binge drinking behaviors and / or become dependent on alcohol. This is a particular concern, as it is suggested that adolescents may be more vulnerable to negative consequences as a result of alcohol use, than their adult counterparts (Petit et al., 2013). One study by Risher et al. (2015) found that binge drinking resulted in functional abnormalities in the frontal lobes in adolescent rats. Additional research is needed in order to determine if this effect is observed in adolescent humans.

Like the frontal lobes, the parietal lobes are also responsible for higher order functioning, in particular, attentional control (Peraza, Cservenka, Herting, & Nagel, 2015). Paulus, Tapert, Pulido and Schuckit (2006) explored the effect of alcohol on the parietal lobes using a working memory task. They found that in comparison to non-drinkers and drinkers who engaged in low level drinking, adults who consumed large amounts of alcohol displayed neural impairments in the parietal lobe and had working memory and sustained attention difficulties. Similarly, Monti et al. (2005) examined the brains of binge drinking and low level drinking adolescents using functional magnetic resonance imaging (fMRI) during completion of moderately difficult spatial working memory tasks. They found that adolescent binge drinkers used additional neural resources from the parietal region to complete tasks, although they had no increase in task performance compared to the adolescent low level drinkers. This suggests that adolescent binge drinkers may need more neural resources to complete tasks, which appears to result in more intense and widespread activation of the brain. It is therefore suggested that adolescents are more likely to experience frontal and parietal lobe deficits as a result of binge drinking than non-binge drinkers.

Doallo et al. (2014) explored the relationship between executive functions and binge drinking in adolescents. They found that adolescent binge drinkers made more errors on a task that requires working memory, the Self-Ordered Pointing Test (SOPT) than adolescent low level drinkers. It was therefore suggested that binge drinking in adolescence is associated with difficulties withholding and monitoring information in working memory. More research into how alcohol influences executive functioning is important in order to further understand how alcohol is influencing cognitive control and executive processes in adolescents.

Males and Binge Drinking

Males tend to be more likely to take behavioural risks than females (Byrnes, Miller & Schafer, 1999). Increased risk taking and impulsivity tends to be related to increased binge drinking (Ferne, Cole, Goudie & Field, 2010). As males are more likely to take risks than females (Isralowitz, Reznik, & Belhassen, 2012; Reilly et al., 1998), it is suggested that they therefore may be more prone to engage in binge drinking behaviours. Moreover, according to a National Drug Strategy Household Survey in Australia, approximately 47% of males aged over 12 engaged in regular binge drinking, whereas approximately 27% of females engaged in binge drinking (Australian Institute of Health and Welfare, 2014). This suggests that males are much more likely than females to engage in binge drinking behaviours.

There also may be a gender difference in terms of vulnerability to cognitive deficits as a result of binge drinking (Scaife & Duka, 2009; Townshend & Duka, 2005). Parada et al. (2012) explored this relationship in male and female binge drinking and non-binge drinking university students. Participants in the binge drinkers group had consumed six or more alcoholic drinks within a single occasion, at least once a month, and participants in the non-binge drinking group had not consumed more than six alcoholic drinks in a single occasion. Participants were matched in terms of gender for each group. Participants completed various tasks (e.g., letter fluency, digit span) that require the use of the prefrontal cortex and executive functions. Males who engaged in binge drinking scored lower on all executive function tasks than female binge drinkers. Male binge drinkers also received lower scores than both male and female non-binge drinkers. Thus males may be more vulnerable to experiencing executive function deficits as a result of binge drinking than females (Parada et al., 2012). It is suggested that males not only

are more likely to engage in binge drinking than females, they also may be at a greater risk of the cognitive impairments and frontal lobe damage associated with binge drinking. Additional research is needed to understand the executive functioning impairments that males experience as a result of binge drinking.

Interference Control and the Flanker/Go-nogo Task

Adults who engage in binge drinking tend to have difficulties completing tasks which require the use of the executive function known as interference control (Rubio et al., 2008). Interference control refers to the ability to respond to stimuli that are relevant to task goals, and to screen out and ignore stimuli that are not relevant to task goals (Brydges et al., 2012). This means that adults who binge drink may find it difficult to maintain attention on tasks, and may be easily distracted by irrelevant information more than adults who do not binge drink. Interference control can be assessed using an experimental paradigm known as the flanker/go-nogo task. The flanker/go-nogo task combines both a flanker task (where a participant is required to respond in a certain way whilst avoiding conflicting stimuli) and a go-nogo task (where a participant is required to withhold their responses to particular stimuli). It is suggested that the flanker component of the task is a measure of interference suppression, which is the attempt to complete a task whilst responding to the target stimuli and ignoring irrelevant and competing information and the go-nogo component of the task is a measure of the complete suppression of behavioural responses (Brydges et al., 2012). It is also suggested that in the flanker/go-nogo task, go incongruent trials (when the target stimuli is surrounded by distractor stimuli facing in opposite directions) may be more sensitive to interference suppression difficulties than go congruent (when the target stimuli is surrounded by distractor stimuli facing in the same direction) trials.

Wilcox, Dekonenko, Mayer, Bogenschutz and Turner (2014) explored the literature of interference control in adults with alcohol use disorder. They found that adults with alcohol use disorder perform more poorly on tasks that require interference control (such as the Stroop task and the flanker task) in comparison to controls. According to Wilcox et al. (2014), adults with alcohol use disorder show higher number of errors on these tasks, as well as higher interference scores. They also found that adults with alcohol use disorder tend to be slower to withhold their responses on tasks such as the Hayling task, which requires individuals to withhold responses (to target words) whilst filtering out irrelevant material (similar distractor words). There is a gap in the literature surrounding whether this effect occurs for individuals who do not necessarily meet diagnostic criteria for alcohol use disorder, but who drink a substantial amount of alcohol.

Ahmadi et al. (2013) explored the relationship between response inhibition and alcohol use, using the go-nogo component of the flanker/go-nogo task. They compared young adult low level drinkers' accuracy on the go-nogo task to young adult binge drinkers' accuracy on the task. They found that young adult binge drinkers made more errors on the go-nogo task in comparison to young adult low level drinkers. These results indicate that the go-nogo task is helpful in detecting particular executive functioning deficits that may occur as a result of heavy alcohol use. There is limited research on the relationship between binge drinking and interference control in adolescents, and how this influences brain electrophysiology. As adolescents who engage in binge drinking appear to be vulnerable to experiencing executive function deficits (Thayanukulvat & Harding, 2015), it is suggested that they also may experience similar difficulties on the flanker/go-nogo

task to adults who binge drink, as well as differences in terms of the electrophysiology of the brain.

Electrophysiology of Interference Control in Binge Drinkers

Electrophysiological activity, such as event-related potentials (ERPs) are the brain's average electrical response to an event and they help tell us the precise timing of specific mental processes. ERPs provide additional information about group differences in cognitive processing whilst individuals complete behavioural tasks (Maurage, Pesenti, Philippot, Joassin & Campanella, 2009).

Commonly investigated ERPs in alcohol and cognition research are the P300 or P3 and the N200 or N2 components. The P3 component has a positive peak of approximately 300 to 600ms and is maximal at the frontal and parietal sites. This component provides information about the specific timing of the various attentional and memory mechanisms that are involved during task processing (Polich, 2007). P3 amplitude is generally reduced when completing tasks that involve greater attentional demands and is increased when engaging in relatively simple tasks that do not require as many competing attentional resources (Polich, 2007). It is generally suggested that more attention is associated with larger P3 amplitude (Sur & Sinha, 2009). Research using the go-nogo component of the flanker/go-nogo task has identified that exploring P3 amplitude at the parietal lobes is useful in providing additional covert information about executive functions, particularly attentional control (Sumich et al., 2008).

In adults, binge drinkers display reduced P3 amplitude compared to non-drinkers on various visual oddball tasks (Crego et al., 2012). Maurage et al. (2009) found that adult binge drinkers had reduced P3 amplitude in comparison to non-drinkers on emotional valence judgment tasks, which involved listening to auditory

stimuli (a male or female voice) expressing negative and positive emotions, and identifying the emotion as happy, sad or neutral. It was suggested that adult binge drinkers appear to have difficulties with the decisional process that occurs in order to terminate cognitive responses, particularly when alternating between multiple tasks. This means that binge drinkers may find it difficult to ignore irrelevant information when working on a task and may have difficulty preventing motor responses. A similar study investigating the P3 amplitude for adult binge drinkers and non-drinkers completing a go/no-go task found similar results (Easdon, Izenberg, Armilio, Yu & Alain, 2005). They also found that P3 amplitude appeared to be reduced for binge drinkers in comparison to non-drinkers. It was suggested that binge drinkers may have impaired ability to maintain their attention and focus on tasks, whilst also filtering out irrelevant information. Additional research is needed to determine if this effect is also observed in adolescents; whether adolescent binge drinkers show reduced P3 amplitude in comparison to adolescent non-binge drinkers.

In addition to the P3 component, the N2 component is another electrophysiological measure which is commonly investigated in alcohol cognition research. The N2 amplitude has a negative peak at approximately 200ms after stimulus onset. The N2 component is associated with various cognitive control functions such as conflict monitoring (Donkers & van Boxtel, 2004). This component also reflects the process involved in the inhibition of responses, and provides additional information in terms of the inhibition of responses to irrelevant stimuli (Easdon et al., 2005; Sokhadze, Stewart, Hollifield, Tasman, 2008).

The N2 amplitude is generally larger when filtering out irrelevant information and withholding responses (Kok, Ramautar, De Ruiter, & Ridderinkhof, 2004). One study explored the relationship between binge drinking and ERPs in

university students (Maurage et al., 2012). They compared the ERPs of binge drinkers and non-binge drinkers on a simple visual oddball task. It was found that binge drinkers had both reduced P3 and N2 amplitudes reflecting difficulties with attention and decision making. Similarly, Crego et al. (2009) found that N2 amplitude was reduced for binge drinkers in comparison to non-binge drinkers. It was suggested that binge drinkers may need additional effort in order to perform tasks adequately. However, there appears to be limited research in the current literature to determine whether binge drinking has an effect on N2 amplitude in adolescents.

The Current Study

Binge drinking appears to be associated with impairments to the frontal lobes of the brain, as well as impairments in various executive functions, such as working memory, attention and interference control (De Bellis et al., 2005; Pfefferbaum, Adalsteinsson, & Sullivan, 2006). This is particularly a concern for adolescents, as the frontal lobes have not yet reached full development, and the brain may be particularly vulnerable to alcohol related harm during the adolescent developmental period (Thayanukulvat & Harding, 2015). Despite this, it is normative in today's society for adolescents to engage in binge drinking behaviours (Roche et al., 2007). The current study will focus on exploring executive functioning deficits in adolescents, as additional research is needed in order to understand more about the potential harms associated with binge drinking in adolescence. It is also suggested that males may be more likely than females to drink alcohol, take risks, and engage in impulsive and binge-drinking behaviours than females (Isralowitz, Reznik, & Belhassen, 2012). Moreover, male adolescents may be more likely than female adolescents to experience cognitive deficits as a result of binge drinking (Parada et

al., 2012). Therefore, the current study will focus on executive functioning in male binge drinkers in comparison to male low level drinkers. The flanker/go-nogo task will be used, in order to further explore the effect of binge drinking on executive functions in adolescent males. In particular, the flanker/go-nogo task will be used to measure interference control and to compare behavioural responses for male adolescent binge drinkers and low level drinkers.

As P3 and N2 amplitude provide additional covert information about individuals' ability to maintain attention on a task with competing attentional demands, as well as information about the ability to inhibit responses to irrelevant material, it is suggested that these electrophysiological measures will provide important information about executive functioning in binge drinking and low level drinking adolescents. The frontal, frontocentral and parietal regions of the brain will be the main area of focus in the current study as this is the area of the brain generally associated with executive functions, as well as the area which research has indicated is most likely to result in damage when an individual engages in binge drinking (Pfefferbaum, Adalsteinsson, & Sullivan, 2006). The P3 and N2 amplitude ERP responses across frontal, central and parietal sites will be recorded whilst participants complete the flanker/go-nogo task, in order to gain additional covert information which cannot be directly observed.

The aim of the current study was to investigate if binge drinking is associated with deficits in behavioral and psychophysiological measures of interference suppression and response inhibition. It was hypothesised that male adolescent binge drinkers would show a significantly higher number of errors on the flanker/go-nogo task and longer reaction times during the flanker task in comparison to low level drinkers. It was expected that this difference would be greater for go incongruent

trials relative to go congruent trials, reflecting poorer levels of executive function and interference suppression for binge drinkers. Moreover, it was also expected that male adolescent binge drinkers would show poorer accuracy on nogo trials than male adolescent low level drinkers, reflecting difficulties with response inhibition.

It was also hypothesised that male adolescent binge drinkers would show significantly reduced N2 amplitude in the frontal and frontocentral electrode sites in comparison to male adolescent low level drinkers. It was expected that this difference would be greater for go incongruent relative to go congruent trials. It was also expected that this difference would be observed for nogo trials. Additionally, it was hypothesised that low level drinkers would show significantly greater N2 amplitude at frontal and frontocentral electrode sites, in comparison to central electrode sites, reflecting greater neural activity in the frontal area of the brain, when inhibiting responses to irrelevant information and attending to relevant stimuli. It was expected that there would be no difference between N2 amplitude for frontal, frontocentral and central electrode sites for binge drinkers, reflecting more widespread activation of the brain when completing a task that requires interference control.

Moreover, it was hypothesised that male adolescent binge drinkers would show significantly reduced P3 amplitude at the frontal and parietal electrode sites in comparison to male adolescent low level drinkers. This difference was suggested to be greater for the go incongruent and nogo relative to the go congruent trials. It was also hypothesised that low level drinkers would show significantly greater P3 amplitude at frontal and parietal electrode sites, in comparison to central electrode sites, reflecting greater neural activity in the frontal area of the brain, making it easier for low level drinkers to maintain attention and focus on tasks. It was expected

that there would be no difference between P3 amplitude for frontal, central and parietal electrode sites for binge drinkers.

Method

Participants

Participants were 22 males aged 15-18 years old ($M=17.3$ years, $SD=0.7$ years). Participants in the 'binge drinkers' group ($n=11$), had consumed more than four standard drinks in one occasion in the 12 months prior to testing (Table 1). Participants in the 'low level drinkers' group had either consumed no alcohol in the 12 months prior to testing ($n=6$), or had never had four or more standard drinks in one occasion over the 12 months prior to testing ($n=5$). Of the participants in the low level drinkers group who had consumed more than one standard drink in the 12 months prior to testing, one participant had consumed 1-3 standard drinks per month, and four participants had consumed alcohol less often than 1-3 days per month. There was no significant difference in age for low level drinkers ($M=17.3$, $SD=0.7$) in comparison to binge drinkers ($M=17.3$, $SD=0.7$), $t(20)=-.13$, $p=.900$. All participants spoke English as their first language and had normal or corrected to normal vision. Additionally, all participants were right handed, with the exception of three (two participants in the binge drinkers group and one in the low level drinkers group) who were left handed. One participant from the 'binge drinkers' group was on psychoactive medication (concerta). All participants were administered the Wechsler Test of Adult Reading (WTAR), in order to obtain an estimate of general Intelligence Quotient (IQ) for each group. A Mann-Whitney U Test revealed no significant difference in the WTAR standard scores of low level drinkers ($Mdn=107.00$, $n=11$) in comparison to binge drinkers ($Mdn=100.00$, $n=11$), $U=55$,

$z=-.362$, $p=.717$. One participant (9.1%) in the ‘binge drinkers’ group had smoked tobacco (2-5 times) within the 12 months prior to participation in the study.

Recruitment was from an existing five year ARC Project Grant examining alcohol use in adolescents (Aiken et al., 2015). Those who had elected that they would like to participate in future studies were invited by email to participate in the current study (see Appendix A). They were invited to click on a web link to a medical screening questionnaire (see Appendix B) to express their interest in participating in the study, and to also ensure eligibility for the study. Exclusion criteria included a history of neurological disorder, illicit drug use, serious head trauma, epileptic seizure or physical condition. Participants had not experienced any serious head injuries. All participants were asked to refrain from drinking any alcohol for 24 hours prior to the experimental session and were reimbursed \$40 for their participation in the study.

Table 1

Means and Standard Deviations (%) of Alcohol Use for Low Level and Binge

Drinkers

	Low Level Drinkers	Binge Drinkers
	(%)	(%)
Any alcohol use in last 12 months	46	100
Frequency of alcohol use in last 12 months		
1-4 days a week	0	18
1-3 days a month	9	36
Less often but at least once	36	46
Never	55	0
Frequency of binge drinking (4 or more standard drinks in one day) in last 12 months		
5 or more days a week	0	0
1-4 days a week	0	0
1-3 days a month	0	9
Less often but at least once	0	91
Never	100	0

Apparatus / Materials

Flanker/go-nogo task. The flanker task was modified from the original Eriksen flanker paradigm (Eriksen & Eriksen, 1974), to include a go/no-go component, as described in Brydges et al. (2012). Each trial consisted of five fish shown on a screen, and participants were asked to focus on the middle target fish. An arrow was displayed on the body of the fish which indicated the fish's direction. Participants were instructed to use their left or right index finger to press a button to

indicate the direction of the middle fish. The task was comprised of three separate conditions, the go congruent, go incongruent and nogo conditions. In the go conditions, all fish were green, however in the go congruent condition all fish were facing in the same direction, whereas in the go incongruent condition, the surrounding four fish were facing in the opposite direction than the middle fish. In the third condition, the nogo condition, four surrounding fish were facing the opposite way to the middle target fish, and were all red, and the participants were asked to withhold their responses. The stimuli were shown on screen in random order, and a central fixation cross was shown for 300ms before each trial. The inter-stimulus interval was 2,000ms. The task consisted of one practice block with eight trials, and one experimental block with 176 trials (44 were nogo trials).

Other measures. The screening questionnaire was administered in order to determine eligibility for the study (see Appendix B). This questionnaire asked questions about the individual's age, handedness, medication, physical health, skin sensitivity, sleep difficulties, tobacco use and alcohol use over the previous 12 months. Participants also completed a brief questionnaire to determine the frequency of (if any) drug use over the 12 months prior to testing (see Appendix B). This was also used to ensure eligibility to the study. The WTAR was also administered in order to gain a general estimate of verbal IQ for each participant. This test measures an individual's ability to read irregular words aloud (Wechsler, 2001). There were 50 items which increased in difficulty.

Electrophysiological Recording

The flanker task was presented using NeuroScan Presentation software and hardware. Electroencephalogram (EEG) data was recorded using 32-channel Quick Cap fitted electrode caps and NeuroScan software and hardware. EEG data was

continuously sampled at a rate of 1000Hz. Data was obtained from 32 electrode sites using the 10-20 electrode placement system (Jasper, 1958). Electrodes were referenced to linked mastoids, with electrode impedances kept below 5k Ω . Electrodes were placed above and below the left eye, and on the outer canthi of each eye which allowed offline correction of eye movement and blink artefacts. Cap fitting took approximately 45 minutes.

The EEG files and the behavioural files were combined offline and filtered using zero-band phase shift filtering at 0.05-30 Hz and at 24dB/Oct. Disruptive eye movements and blinks were reduced using a baseline correction and an ocular artefact reduction process. Correct trials for each stimulus type were averaged for a 1s period beginning 100ms prior to stimulus onset. The artefact reduction procedure used values with a 100 μ V high voltage cut off to a -100 μ V low voltage cut off. The posterior N2 and P3 amplitudes were determined from grand averaged waveforms, and defined as the minimum and maximum voltage, which was between 210-300ms and 300-550ms after stimulus onset, respectively.

Procedure

This research was permitted by the Human Research Ethics Committee (HREC) of the University of Tasmania (see Appendix C). Participants were selected from the existing ARC study on the basis of their reported drinking engagement and invited to complete the online screening questionnaire. Participants who met eligibility requirements were then invited to the University of Tasmania Psychology Research Centre to participate in the experimental session. All participants were given an information sheet with details about the study, and written parental and participant consent was obtained before participants took part in the study (see Appendices D and E). When participants entered the research lab, the Quick Cap

was fitted and they were then seated 60 centimeters in front of a computer screen, to complete the computer tasks. The participants completed three separate computer tasks in a set order, however only the flanker/go-nogo task will be reported. They were instructed to respond as accurately and as quickly as possible. All participants completed a practice block of eight trials before completing one experimental block with 176 trials. The experimental session was approximately two hours in total for each participant.

Design and Data Analysis

The current study used a 2(Group: Binge, Low-level drinkers) x 3(Trial type: go congruent, go incongruent, nogo) mixed ANOVA design to analyse data (see Appendix F). The behavioural dependent measures were reaction time (ms; go conditions only) and accuracy (percentage of correct responses) for the flanker/go-nogo task. The psychophysiological dependent measures were the mean peak amplitude of the P3 and N2 components. There was an additional independent variable of Site for the N2 amplitude (Site: FZ, FCZ and CZ electrode sites) and the P3 amplitude (Site: FZ, PZ, CZ electrode sites) ERP dependent variables (see Appendix F).

Significant main effects and interactions were assessed using pairwise comparisons, with Greenhouse-Geisser corrections used as appropriate. Significance levels were set at $p < .05$. Bonferroni corrections were used when exploring simple effects that involved three or more comparisons, in order to control for Type 1 error. For these analyses, significance values were set at $p < .017$ ($0.05/3 = .017$).

In addition to peak amplitude analyses, difference waveforms were constructed according to established methodology from Brydges et al. (2012) who used a similar flanker/go-nogo task design. The go congruent trial was subtracted

from the go incongruent trial to create the Incongruent/Congruent Go difference waveform condition (IC Go difference). The go incongruent trial was subtracted from the nogo trial to create the No Go Incongruent difference waveform condition (NGI difference). The mean amplitude of the difference waveforms was calculated between 0-450ms and analysed at FZ, FCZ and CZ electrode sites, using a mixed ANOVA.

Results

Reaction Time

Mean Reaction Time for drinking group and task condition are displayed in Table 2. There was a significant overall main effect of Trial on Reaction Time, $F(1,20)=250.80$, $p < .001$, $\eta_p^2 = .93$. Reaction Time was significantly slower on incongruent ($M=481.84$, $SD=45.53$) than congruent ($M=393.48$, $SD=28.89$) go trials. There was no significant interaction between Group and Congruency, $F(1, 20)=.11$, $p=.748$, $\eta_p^2=.01$. The main effect of Group was also non-significant, $F(1, 20)=.04$, $p=.836$, $\eta_p^2 < .01$.

Accuracy

The mean accuracy (percentage of correct responses) for each group and task condition are displayed in Table 2. There was a significant overall main effect of Trial, $F(1, 20)=73.18$, $p < .001$, $\eta_p^2 = .79$, indicating that accuracy was significantly lower for go incongruent trials ($M=73.86$, $SD=12.81$) compared to both go congruent ($M=97.16$, $SD=2.59$, $p < .001$) and nogo trials ($M=96.59$, $SD=3.89$, $p < .001$). There was a non-significant interaction between drinking group and congruency, $F(1, 20)=.28$, $p=.600$, $\eta_p^2=.01$. The main effect of drinking group was also non-significant, $F(1, 20)=.38$, $p=.543$, $\eta_p^2=.02$.

Table 2

Mean (SD) and 95% CI for Reaction Time and Accuracy for Low Level and Binge Drinkers

	Low Level Drinkers		Binge Drinkers	
	<i>n</i> =11		<i>n</i> = 11	
Reaction Time	<i>M</i> (<i>SD</i>)	95% CI	<i>M</i> (<i>SD</i>)	95% CI
Go Congruent	396.03 (21.95)	[377.49, 414.57]	390.93(35.45)	[372.38, 409.47]
Go Incongruent	482.58 (36.08)	[453.24, 511.91]	481.10(55.23)	[451.77, 510.44]
Accuracy (%)				
Go Congruent	97.83 (2.35)	[96.22, 99.44]	96.49 (2.76)	[94.88, 98.10]
Go Incongruent	75.41 (13.05)	[67.22, 83.61]	72.31 (13.01)	[64.12, 80.51]
No go	96.49 (4.36)	[93.98, 98.99]	96.69 (3.57)	[94.19, 99.20]

Electrophysiological Data

Grand mean averaged waveforms and difference waveforms for participants in the low level drinking group and binge drinking groups are displayed in Figure 1 and 2 respectively.

N2 amplitude. There was a significant main effect of Trial type, $F(2, 40)=6.24$, $p=.004$, $\eta_p^2=.24$, where N2 amplitude was significantly greater in the nogo condition ($M=-4.49$, $SD=5.72$, 95% CI [-7.04, -1.94]) than both the go congruent ($M=-2.59$, $SD=4.60$, 95% CI [-4.62, -.55], $p=.006$) and the go incongruent conditions ($M=-2.65$, $SD=4.46$, 95% CI [-4.64, -.66], $p=.013$). There was no significant difference between the go congruent and go incongruent conditions ($p=.905$). There

were no significant main effects of Site, $F(2, 40)=2.12, p=.134, \eta_p^2=.10$, or Group, $F(1, 20)=.14, p=.714, \eta_p^2=.01$. Moreover, there was no significant interaction between Trial type and Group, $F(2, 40)=0.65, p=.526, \eta_p^2=.03$, or between Trial type, Site and Group, $F(2, 40)=0.31, p=.874, \eta_p^2=.02$.

There was a significant interaction between Group and Site, $F(2, 40)=4.27, p=.021, \eta_p^2=.18$, indicating that N2 amplitude differed for low level and binge drinkers at different electrode sites (see Figure 3). For low level drinkers, there was a significant main effect of Site, $F(2, 20)=7.35, p=.004, \eta_p^2=.42$. Pairwise comparisons ($p<.017$, Bonferroni corrected) indicated greater N2 amplitude at the FZ electrode site ($M=-3.67, SD=5.14, p=.014$) compared to the CZ ($M=-1.97, SD=4.08$) electrode site, with a trend for greater N2 amplitude at FZ than at FCZ ($M=-2.98, SD=4.97, p=.022$) and no significant difference between the FCZ and CZ electrode sites ($p=.090$). For binge drinkers, there was a non-significant main effect of Site on N2 amplitude, $F(2, 20)=.42, p=.670, \eta_p^2=.04$. There were no significant main effects of Group at the FZ, $F(1, 20)=.25, p=.875, d<.01$, FCZ, $F(1, 20)=.16, p=.698, d=.20$, and CZ, $F(1, 20)=.81, p=.378, d=.41$, electrode sites.

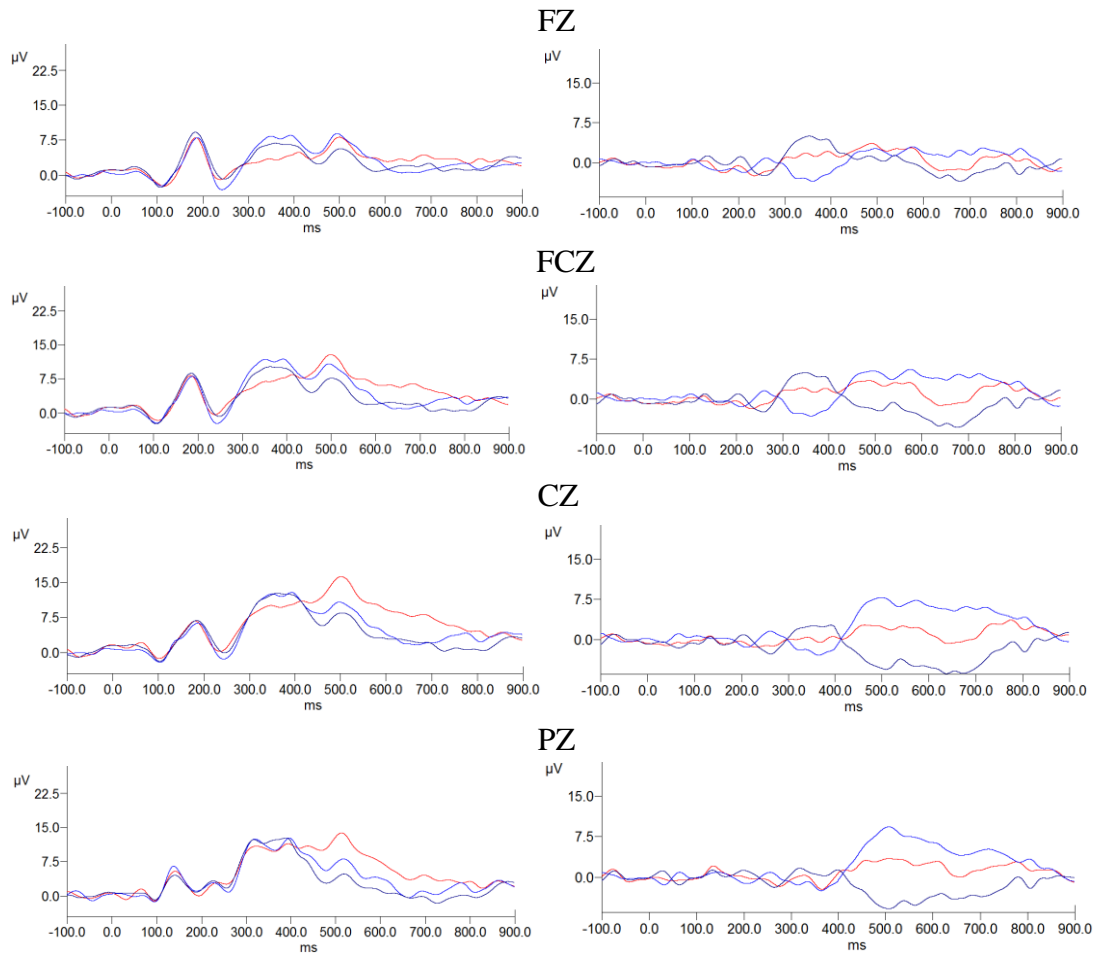


Figure 1. Grand mean averaged waveforms (left hand side) and difference waveforms (right hand side) for low level drinkers. Left hand side: Grand mean averaged ERP in response to go congruent (dark blue), go incongruent (red) and nogo (light blue) trials. Right hand side: Grand mean averaged difference waveforms displayed as the nogo – go incongruent (dark blue), nogo – go congruent (red), go incongruent – go congruent (light blue) waveforms.

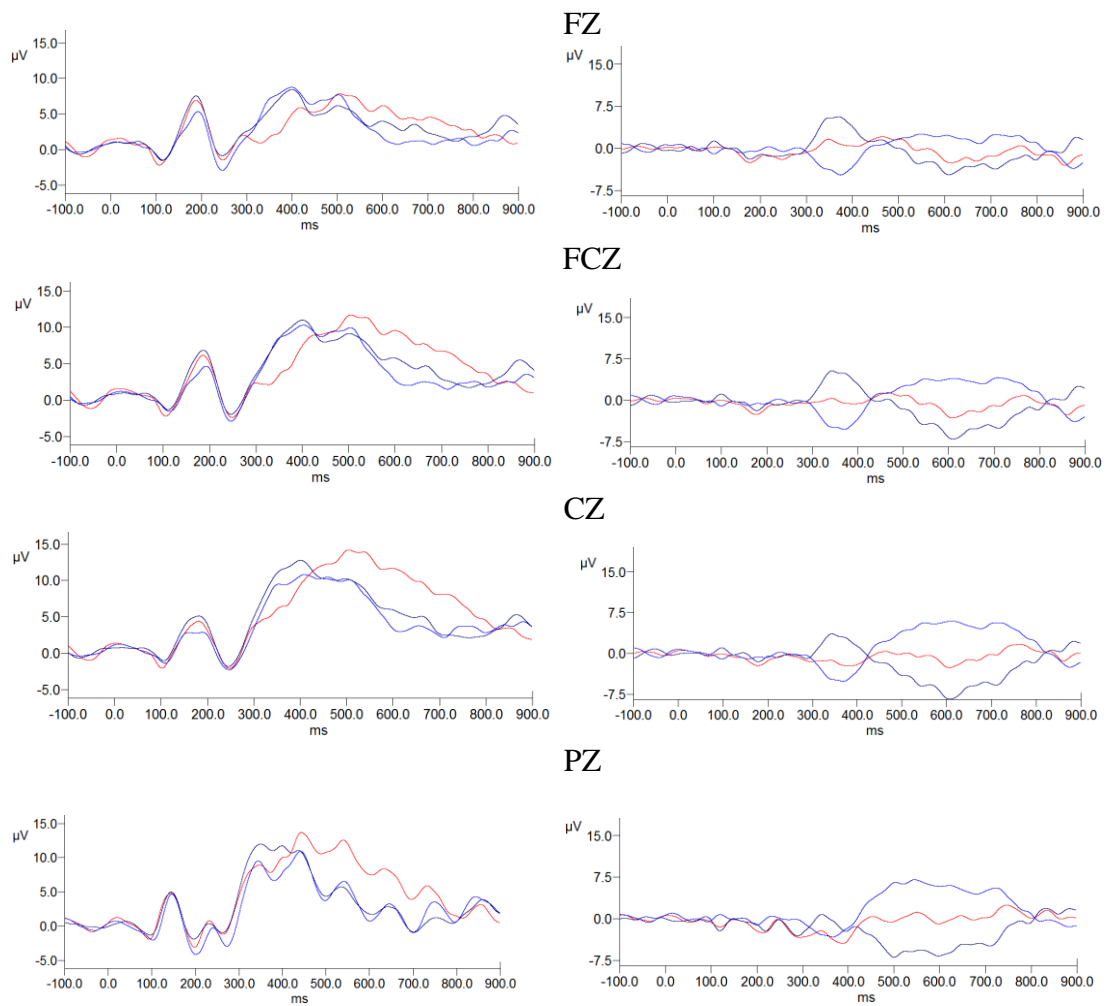


Figure 2. Grand mean averaged waveforms (left hand side) and difference waveforms (right hand side) for binge drinkers. Left hand side: Grand mean averaged ERP in response to go congruent (dark blue), go incongruent (red) and nogo (light blue) trials. Right hand side: Grand mean averaged difference waveforms displayed as the nogo – go incongruent (dark blue), nogo – go congruent (red), go incongruent – go congruent (light blue) waveforms.

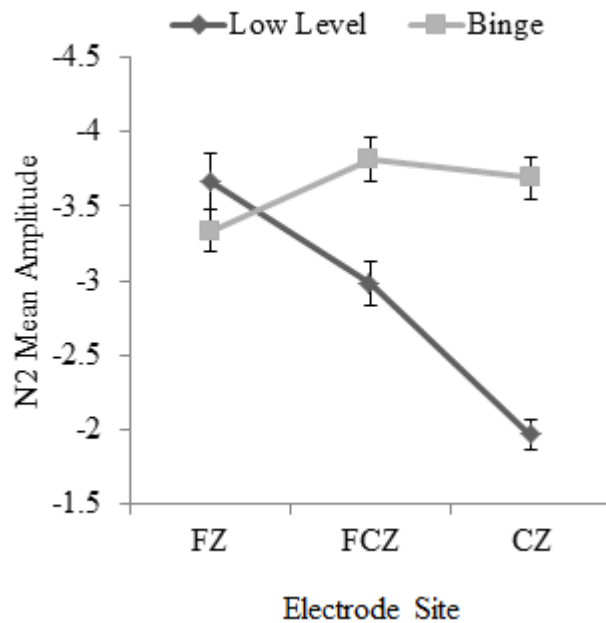


Figure 3. Mean peak N2 amplitude for low level and binge drinkers at frontocentral (FZ, FCZ, CZ) electrode sites. Error bars represent 95% confidence.

There was also a significant interaction between Site and Trial, $F(2, 40)=3.16$, $p=.018$, $\eta_p^2=.14$, (see Figure 4). Breakdown analyses revealed that the main effect of Site was non-significant for go congruent ($p=.486$), and go incongruent ($p=.354$) trials, but approached significance for no go trials ($p=.042$). For no go trials, there was a trend ($p<.017$, Bonferroni corrected) indicating greater N2 amplitude at the FZ ($M=-5.16$, $SD=6.04$) compared to the CZ ($M=-3.74$, $SD=5.16$) electrode sites ($p=.041$). For no go trials, there was no significant difference between FCZ ($M=-4.56$, $SD=6.03$) and FZ ($p=.136$) or between FCZ and CZ ($p=.056$) electrode sites. There was a significant main effect of Trial type on N2 amplitude at the FZ electrode site, $F(2, 40)=9.31$, $p<.001$, $\eta_p^2=.32$. Pairwise comparisons ($p<.017$, Bonferroni corrected) revealed that at the FZ site, there was greater N2 amplitude for no go trials ($M=-5.16$, $SD=6.05$) compared to go congruent ($M=-2.51$, $SD=4.55$, $p<.001$) and go incongruent ($M=-2.83$, $SD=4.50$, $p=.007$) trials.

At the FZ site, there was no significant difference between go congruent and go incongruent trials ($p=.577$). The main effects of Trial type trended towards significance ($p<.017$, Bonferroni corrected) at the CZ, $F(2, 40)=3.36$, $p=.045$, $\eta_p^2=.14$, and FCZ, $F(2, 40)=4.51$, $p=.017$, $\eta_p^2=.18$, electrode sites. At the FCZ site, there was a trend ($p<.017$, Bonferroni corrected) for greater N2 amplitude for nogo trials ($M=-4.56$, $SD=6.05$) compared to go congruent ($M=-2.83$, $SD=4.83$, $p=.022$) and go incongruent ($M=-2.80$, $SD=4.55$, $p=.026$) trials. There was no significant difference between go incongruent and go congruent trials ($p=.952$) at the FCZ site. Additionally, at the CZ site, there was also a trend ($p<.017$, Bonferroni corrected) for greater N2 amplitude for nogo trials ($M=-3.74$, $SD=5.16$) compared to go congruent ($M=-2.42$, $SD=4.55$, $p=.046$) and go incongruent ($M=-2.33$, $SD=4.50$, $p=.026$) trials. At the CZ site, there was no significant difference between go incongruent and go congruent trials ($p=.883$).

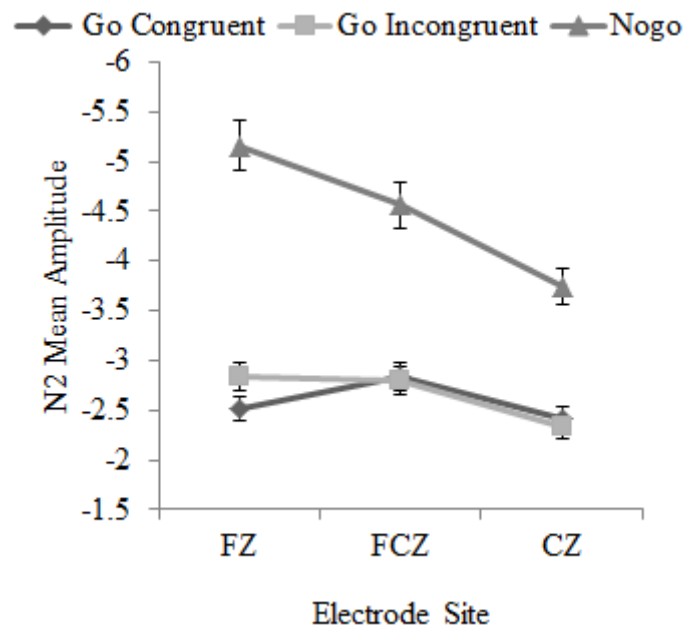


Figure 4. Mean peak N2 amplitude for go congruent, go incongruent and nogo incongruent trials across FZ, FCZ and CZ electrode sites. Error bars represent 95% confidence.

N2 mean amplitude, difference waveforms. A mixed ANOVA was conducted to assess the impact of Trial type (IC Go difference condition and NGI difference condition) across different electrode Sites (FZ, FCZ and CZ) on N2 amplitude difference waveform for low level and binge drinkers. There was a significant main effect of Trial type, $F(1, 20)=7.38$, $p=.013$, $\eta_p^2=.27$, where mean N2 amplitude was significantly greater in the NGI difference condition ($M=-1.90$, $SD=2.86$, 95% $CI [-3.18, -.62]$) than the IC Go difference condition ($M=-.07$, $SD=2.53$, 95% $CI [-1.19, 1.06]$). There was a significant main effect of Site, $F(2, 40)=4.24$, $p=.021$, $\eta_p^2=.18$, where there was a trend ($p<.017$, Bonferroni corrected) indicating that N2 amplitude was greater at the FZ site ($M = -1.49$, $SD = 2.06$, 95% $CI [-2.41, -.56]$) in comparison to the FCZ site ($M=-.85$, $SD=2.39$, 95% $CI [-1.91, .22]$, $p=.025$). There was also a trend for greater N2 amplitude at the FZ site in comparison to the CZ site ($M=-.62$, $SD=.2.53$, 95% $CI [-1.74, .50]$, $p=.048$). There was no significant difference between the FCZ and the CZ sites ($p=.230$).

There was a non-significant main effect of Group, $F(1, 20)=1.01$, $p=.326$, $\eta_p^2=.05$. Additionally, there were non-significant interactions between Site and Group, $F(2, 40)=.23$, $p=.795$, $\eta_p^2=.01$, Trial type and Site, $F(2, 40)=2.39$, $p=.105$, $\eta_p^2=.11$, Trial type and Group, $F(1, 20)=.42$, $p=.523$, $\eta_p^2=.02$, and between Trial type, Site and Group, $F(2, 40)=.36$, $p=.702$, $\eta_p^2=.02$.

P3 amplitude. There was a significant main effect of Site, $F(2, 40)=29.95$, $p<.001$, $\eta_p^2=.60$, where P3 amplitude was significantly greater at the CZ ($M=15.93$, $SD=6.00$, 95% $CI [13.27, 18.60]$, $p<.001$) and the PZ electrode sites ($M=15.30$, $SD=4.74$, 95% $CI [13.20, 17.40]$, $p<.001$) than the FZ site ($M=10.85$, $SD=4.97$, 95% $CI [8.63, 13.07]$). There were non-significant main effects of Trial type, $F(2, 40)=2.40$, $p=.104$, $\eta_p^2=.11$, and Group, $F(1, 20)=.06$, $p=.802$, $\eta_p^2<.01$. Moreover,

there were non-significant interactions between Trial type and Group, $F(2, 40)=1.37$, $p=.265$, $\eta_p^2=.06$, Group and Site, $F(2, 40)=1.12$, $p=.335$, $\eta_p^2=.05$, and Trial type, Site and Group, $F(4, 80)=1.20$, $p=.317$, $\eta_p^2=.06$.

There was a significant interaction between Site and Trial type, $F(2, 40)=7.42$, $p<.001$, $\eta_p^2=.27$ (see Figure 5). There were significant main effects of Trial type on P3 amplitude at the CZ, $F(2, 40)=4.38$, $p=.019$, $\eta_p^2=.18$, and FZ electrode sites, $F(2, 40)=3.63$, $p=.036$, $\eta_p^2=.15$, but not at PZ, $F(2, 40)=2.97$, $p=.063$, $\eta_p^2=.13$. Pairwise comparisons ($p<.017$, Bonferroni corrected) revealed that P3 amplitude at the CZ site was greater for go incongruent trials ($M=17.49$, $SD=8.02$) than the go congruent ($M=14.95$, $SD=6.00$) trials ($p=.013$). There was no significant difference between nogo trials ($M=15.36$, $SD=4.78$) and go incongruent ($p=.067$) or go congruent trials ($p=.610$). Moreover, at the FZ site, there was a trend toward significant difference ($p<.17$, Bonferroni corrected), where the nogo trials ($M=12.06$, $SD=5.82$) showed greater P3 amplitude than congruent go ($M=9.86$, $SD=4.83$) trials ($p=.022$). At the FZ site there were non-significant differences between go incongruent trials ($M=10.63$, $SD=5.44$) and both congruent go ($p=.328$) and nogo trials ($p=.094$).

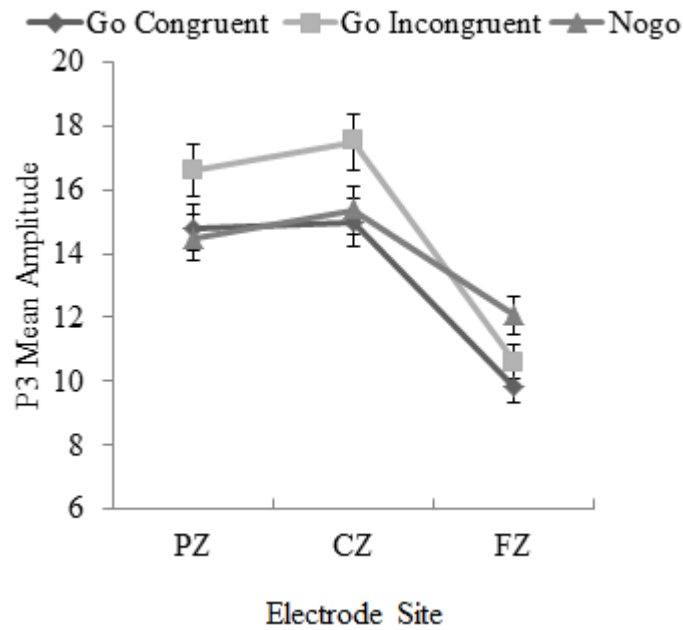


Figure 5. Mean peak P3 amplitude for go congruent, go incongruent and nogo incongruent trials across PZ, CZ and FZ electrode sites. Error bars represent 95% confidence.

There were significant main effects of Site on P3 amplitude for the nogo, $F(2, 42)=8.57, p=.001$, go incongruent, $F(2, 42)=33.56, p<.001$, and go congruent, $F(2, 42)=26.13, p<.001$, trial types. Pairwise comparisons ($p<.017$, Bonferroni corrected) for each trial type are displayed in Table 3. For nogo, go incongruent and go congruent trials, P3 amplitude was greater at the CZ electrode site in comparison to the FZ electrode site. Additionally, for go incongruent and go congruent trials, P3 amplitude was greater at the PZ electrode site in comparison to the FZ electrode site. For nogo trials there was a trend, indicating that P3 amplitude was greater at the PZ electrode site in comparison to the FZ electrode site. For nogo, go incongruent and go congruent trials there was no significant difference between PZ and CZ electrode sites.

Table 3

Pairwise Comparisons for each Trial Type at each Electrode Site (PZ, CZ, FZ)

	<i>M (SD)</i>	Significant value (<i>p</i>)
Nogo trials		
PZ vs CZ	14.48(4.70) vs 15.36(4.79)	.176
FZ vs CZ	12.06(5.82) vs 15.36(4.79)	<.001***
PZ vs FZ	14.48(4.70) vs 12.06(5.82)	.037**
Go incongruent trials		
PZ vs CZ	16.61(6.45) vs 17.49(8.02)	.262
FZ vs CZ	10.63(5.42) vs 17.49(8.02)	<.001***
PZ vs FZ	16.61(6.45) vs 10.63(5.42)	<.001***
Go congruent trials		
PZ vs CZ	14.81(4.49) vs 14.95(6.02)	.865
FZ vs CZ	9.86(4.82) vs 14.95(6.02)	<.001***
PZ vs FZ	14.81(4.49) vs 9.86(4.82)	<.001***

Note: ** $p < .05$. *** $p < .001$.

Discussion

The aim of the current study was to investigate if binge drinking was associated with deficits in behavioural and psychophysiological measures of interference suppression and response inhibition. The hypothesis that male adolescent binge drinkers would show a significantly higher number of errors on the flanker/go-nogo task and longer reaction times during the flanker task in comparison to low level drinkers was not supported. There were no differences on reaction time and accuracy for binge and low level drinkers. Therefore, the hypothesis that the difference would be greater for the go incongruent trials relative to the go congruent

trials, and that binge drinkers would show poorer accuracy on nogo trials than low level drinkers, was also not supported.

Contrary to expectations, male adolescent binge drinkers did not show significantly reduced N2 amplitude when completing the flanker/go-nogo task in comparison to male adolescent low level drinkers. Therefore, there was also no difference in N2 amplitude across trials for low level and binge drinkers. As expected, low level drinkers showed significantly greater N2 amplitude at frontal electrode sites in comparison to central electrode sites. Additionally, as expected there was no difference between N2 amplitude for frontal, frontocentral and central electrode sites for binge drinkers. The hypothesis that male adolescent binge drinkers would show significantly reduced P3 amplitude at frontal electrode sites in comparison to male adolescent low level drinkers was not supported. Contrary to expectations, there was no difference across trials. Additionally, the hypothesis that low level drinkers would show significantly greater P3 amplitude at frontal electrode sites, in comparison to central and parietal electrode sites was not supported. As expected, there was no difference between P3 amplitude for frontal, central and parietal electrode sites for binge drinkers.

The greater N2 amplitude for frontal electrode sites in comparison to the central electrode sites for low level drinkers indicates that low level drinkers may predominately use electrophysiological resources from within the frontal lobes to inhibit their responses and filter out irrelevant stimuli. It is suggested that binge drinkers recruit central resources as well as resources from the frontal lobes. As the frontal lobes of the brain are responsible for executive functions such as interference suppression and response inhibition (Russo, 2003), it may be more helpful for individuals to specifically use resources from the frontal lobes when completing

tasks that require executive functioning. It is suggested that these neural processes are being used to facilitate low level drinkers' ability to complete the flanker/go-nogo task. This raises questions however, as to why there was not a similar effect for binge drinkers. This also raises questions about why there was not a difference in terms of the reaction time and accuracy, given that there was a difference in terms of electrophysiological function. Additionally, it also raises questions about what the results of the present study means in terms of response inhibition and interference suppression for adolescent male binge drinkers and low level drinkers.

Binge drinking and Interference Control On the Flanker/Go-nogo Task

The current results found that there was no difference between binge drinkers and low level drinkers in reaction time and accuracy on the flanker/go-nogo task, reflecting no behavioural differences in terms of interference control. The literature review by Wilcox et al. (2014) found interference control difficulties in adults with alcohol use disorder. These researchers did not explore the differences of reaction time and accuracy on interference control tasks for individuals who consume large amounts of alcohol but do not meet diagnostic criteria for alcohol use disorder. Additionally, they also did not review the literature on alcohol use in adolescents. The current results may suggest that adolescent binge drinkers' difficulties with interference control may not be as profound as the difficulties with interference control that individuals with alcohol use disorder experience. Alternatively, the results of the current study may suggest that there is no difference between binge drinkers' and low level drinkers' ability to filter out irrelevant information on tasks.

The results of the current study found no difference in terms of accuracy on the go-nogo component of the flanker/go-nogo task, reflecting no difference of low level and binge drinking in the ability to inhibit behavioural responses. A study by

Easdon et al. (2005) found different results. They found that low and moderate adult drinkers displayed more errors on the go-nogo task than non-drinkers. This study separated participants into three groups; low level, moderate and non-drinkers, whereas the current study separated participants into two groups: low level and binge drinkers. The Easdon et al. (2005) study also provided participants with alcohol that placed them in a drinking condition, rather than exploring how much alcohol participants had consumed over a period of time. It is suggested that the effects of drinking large amounts of alcohol may be more pronounced for individuals who are currently intoxicated. The results from the present study may suggest that deficits in interference control may be subtle when there are no additional challenges; however, if the task becomes more difficult (i.e. when the individual engaging in the task is intoxicated) difficulties in interference control may become more apparent.

Binge Drinking and Electrophysiological Responses

The results of the present study found that male adolescent binge drinkers did not show significantly reduced N2 amplitude or P3 amplitude in comparison to male adolescent low level drinkers. Research by Maurage et al. (2012) found conflicting results, and found that adult binge drinkers had reduced N2 amplitude and P3 amplitude in comparison to non-drinkers. These researchers used a tightly controlled experimental design and explored ERP differences on a visual oddball task, across four groups. They classified these groups as nondrinkers, daily drinkers, low binge drinkers and high binge drinkers, using strict criteria that took into consideration alcohol dose per drinking session, number of drinking sessions per week, the speed of drinking per hour, and number of alcoholic drinks consumed per week. Although they defined binge drinking as being more than 5 standard drinks in an hour which is similar to the present study, they may have found different results as they used a

much more tightly controlled experimental design, using many different criteria to define binge drinking. It is possible that the present study may have found similar results if the criteria for binge drinking had of been more strict. Additionally, Maurage et al. (2012) used a slightly different task to the flanker/go-nogo task, which looked at emotional processing and decision making rather than interference control. This suggests that binge drinkers may display differences from low level drinkers in terms of their electrophysiology (N2 and P3 amplitude) when using other executive functions such as decision making and filtering emotional processing information rather than interference suppression and response inhibition. Alternatively, Maurage et al. (2012) obtained a total of 80 participants in their overall sample, which is much larger than the 22 participants recruited in the present study. It is possible that the discrepancy in the results of the Maurage (2012) study in comparison to the results of the present study may be a function of the low experimental power in the current study.

The results from the current ERP analyses found that N2 amplitude was greater in the frontal areas of the brain in comparison to the central areas of the brain for low level drinkers. This effect was not found for binge drinkers. This result suggests that there are differences in terms of electrophysiology for low level drinkers in comparison to binge drinkers in the ability to filter irrelevant information. It is suggested that this difference is not large enough to show variance in terms of binge drinkers' ability to complete the flanker task in comparison to low level drinkers. As low level drinkers had greater N2 amplitude at frontal electrode sites compared to more central electrode sites, it is suggested that they use more neural resources in the frontal areas of their brain when completing tasks that require the use of executive functions such as interference control. It can also be suggested that

binge drinkers may use resources from other areas of the brain to help them to focus on one task whilst ignoring conflicting demands, than low level drinkers. This is consistent with findings from Monti et al. (2005), who found that adolescent binge drinkers used additional neural resources from the parietal region of the brain when completing moderately difficult working memory tasks. Similar to the results from the current study, they also found that binge drinkers did not display an increase in task performance in comparison to low level drinkers. This suggests that adolescent binge drinkers may use additional resources when completing various executive functioning tasks that require working memory as well as interference control, with no increased task performance.

As the frontal lobes generally control various executive functions (Russo, 2003), the current results suggest that individuals who have not engaged in binge drinking patterns may make better use of neural resources (which allows for easier executive functioning) than those who do engage in binge drinking. It is suggested that using neural resources in the frontal lobes may be more adaptive when requiring executive functions than using neural resources in other areas of the brain (Bryan & Luszcz, 2000). These subtle differences may not be enough to impair adolescent binge drinkers' ability to filter out irrelevant material and withhold responses on everyday tasks; however it is suggested that binge drinkers may require more neural resources to perform tasks at the same level as low level drinkers. In light of the Easdon et al. (2005) study as mentioned above, these deficits may also become more apparent when an individual is tired, stressed or influenced by alcohol. This effect occurs across a range of contexts, including mild traumatic brain injury (TBI). When individuals have experienced mild TBI they tend to perform more poorly when they are fatigued than when they are not fatigued on tasks that require working memory

(Johansson, Berglund, & Rönnbäck, 2009). It is suggested that this is because the brain is required to work overtime to complete tasks, which may be more difficult if an individual is fatigued or stressed. The results from the current study indicate that this may be the case for adolescent males who engage in regular binge drinking.

Additionally the current results also show that alcohol use appears to begin to make changes to aspects of the brain for binge drinkers during adolescence. The literature review by Wilcox et al. (2014) found that adults who meet criteria for alcohol use disorder show differences in terms of their electrophysiological responses as well as differences in their ability to perform executive functioning tasks. The results from the present study may indicate that these differences may occur if adolescent binge drinkers continue to engage in binge drinking into adulthood. Thus, reflecting a possible predisposition to cognitive difficulties in adulthood for individuals who begin binge drinking patterns in adolescence. More research of the longitudinal nature is needed in order to confirm this.

Limitations and Future Research

It is important to consider the results of this study in the context of its limitations. A potential limitation of the present study may be the criteria used to define binge drinking. Whilst many previous studies tend to classify binge drinking as consuming approximately 4-6 standard drinks in one occasion, there is a lack of consensus around other criteria such as time of consumption and how many times an individual can engage in binge drinking behaviours before it begins to have damaging effects on the structure and function of the brain (Beccaria, Petrilli & Rolando, 2015). One study by Renner, O'Dea, Sheehan and Tebbutt (2015) described binge drinking as consuming six or more standard drinks during one session either weekly or approximately daily, whereas another study by Hutter,

Lawton, Pals, O'Connor and McEachan (2015) described binge drinking as drinking more than twice the maximum recommended alcoholic drinks per day in one occasion. The lack of specific guidelines surrounding binge drinking makes it difficult to make conclusions about the effects of binge drinking in adolescents, as well to make comparisons between studies. It is possible that the definition of binge drinking in the current study (i.e. having four or more standard drinks in one occasion in a 12 month period) may be too little to make a difference from low level drinkers in terms of binge drinking and damaging effects of the brain. It could be debateable that three standard drinks on one occasion in a 12 month period may have similar effects to the brain as four standard drinks in the same time frame. Additionally, the participants from the current study who were classified as binge drinkers had only engaged in binge drinking at maximum between 1-3 days per month. This is relatively low frequency of binge drinking, which may have been somewhat different to the frequency of binge drinking observed in other studies exploring the effects of heavy alcohol use in executive functioning. It is proposed that additional research is needed in order to construct an accepted definition of what classifies binge drinking. The present study may have benefited from a bigger discrepancy between low level and binge drinking and stricter criteria for binge drinking. However, this may not have had a large impact on the results of the current study, as the definition of binge drinking in the current study was tied to the current Australian health policy definition of binge drinking, which is 5-6 standard drinks in a single occasion (NHMRC, 2009). This implies that the results from the present study can still be generalised to Australian adolescents.

Another limitation of the current study was that only the flanker/go-nogo task was used to measure executive functioning. The flanker/go-nogo task measures

interference control and response inhibition and does not measure other aspects of executive function. Research indicates that other aspects of executive function such as planning and decision making may be related to binge drinking (Mullan, Wong, Allom & Pack, 2011; Townshend et al., 2014). It is therefore difficult to make general conclusions about executive functioning in binge drinkers where there are other important areas of executive function that have not been explored within this study. Further research is needed in order to determine if binge drinking appears to make a difference in all areas of executive function or if this difference is specific to certain aspects of executive functioning such as interference control and response inhibition.

A possible limitation of the current study was the limited number of participants. Unfortunately, after controlling for various standard ERP exclusionary criteria, the amount of participants left in the study was minimal. In previous ERP studies exploring the effect of binge drinking on cognition, many more participants have been used (Maurage et al., 2009). The present study may have found a greater difference between binge drinkers and low level drinkers in terms of executive function if there were more participants included in the study and greater statistical power. More research is needed exploring executive functioning for binge drinkers in comparison to low level drinkers, to further confirm whether there is no difference between binge drinking and executive functioning or whether the current findings were as a result of the small sample size.

Additionally, research has suggested that low level alcohol consumption (ie, consuming less than 4 standard drinks in one occasion) is related to greater amounts of cognitive difficulties in comparison to not drinking at all (Ganguli et al., 2005). The current study combined low level drinkers (less than 4 standard drinks in one

occasion across a 12 month period) with non-drinkers (participants who had consumed no alcohol in the previous 12 months). A study by Easdon et al. (2005) placed low level drinkers in a separate group to non-drinkers, and found that low level drinkers made more errors withholding responses on a go-nogo task than non-drinkers. As the current study combined both non-drinkers and low level drinkers in one group this may have had an impact on the results of the current study, as low level drinkers may have made more errors than non-drinkers. It is suggested that the current study may therefore be overlooking the potential for differences in executive functioning for low level drinkers in comparison to non-drinkers. As the sample size was so low in the current study, it would not have been appropriate to explore whether this is the case when splitting up the low level drinkers group. There appears to be a gap in the literature around how alcohol consumption influences executive functioning in low level drinkers, and this is an area that may be beneficial to explore with future research.

It is also important to note that most research on alcohol use, including the current study, is cross-sectional rather than longitudinal. This makes it difficult to determine whether between group differences are related to pre-existing differences in executive functions and/or damage to particular areas of the brain, or the impact of excessive drinking. More longitudinal research is needed in order to further understand the results of the present study. However, the results from the present study still provide useful information about the differences in executive functioning between binge drinking and low level drinking in adolescent males.

Practical Implications

Despite the limitations listed previously, the current study offers further understanding of the link between binge drinking and executive functioning. The

current study raises important questions about binge drinking in adolescence, and whether binge drinking is harmful for adolescents. The findings suggest that binge drinkers appear to recruit neural resources from other areas of the brain when using executive functions, whereas low level drinkers appear to recruit neural resources from the frontal lobes. It is suggested that binge drinking may impact on the developing brains of adolescents, however this impact appears to be a more subtle difference that may not be observable in terms of having difficulties completing tasks. This is relatively alarming, as adolescent males who binge drink are not likely to experience any direct difficulties when completing cognitive tasks, which may make them more likely to continue to binge drink into adulthood. Additionally, the results from the present study suggest that male adolescent binge drinkers may be more vulnerable to the effects of fatigue, stress or other impairments when completing tasks which require executive functions. This suggests that male adolescent binge drinkers may perform more poorly on executive function tasks when fatigued or stressed. Further research is needed in order to confirm this.

Evidence from previous research suggests that cognitive differences between low level drinking and binge drinking patterns are more profound for adults who meet diagnostic criteria for alcohol use disorder, as both differences in brain electrophysiology is observed as well as differences in executive functioning tasks (Wilcox et al., 2014). It may therefore be possible that cognitive impairment as a result of binge drinking may begin in adolescence and progress into adulthood if an individual continues to binge drink. This highlights the importance of early intervention for adolescents and the need to provide education to adolescent males about the risks associated with binge drinking and how consequences of binge drinking can be covert rather than easily observable. It also may be particularly

important to encourage and emphasise to adolescents who already engage in binge drinking the importance of low level drinking patterns rather than binge drinking. Additionally, it may also be very important to explain to parents of males in adolescence the potential harm of binge drinking patterns. It may be helpful to discuss with parents ways in which they can monitor their adolescent children in terms of how much they are drinking and how to restrict the amount of alcohol that their children are drinking. It may also be beneficial for parents to reinforce and reward low level drinking patterns in controlled environments, if they are unable to deter their children from engaging in alcohol use. The results from the current study may also suggest that it may be particularly important for psychologists working with young males who engage in binge drinking to prioritize helping the client to reduce their alcohol use, and to provide psycho education about the risks associated with binge drinking.

The current study adds to existing research that suggests that binge drinking is harmful in adults (Pfefferbaum, Adalsteinsson, & Sullivan, 2006) by providing additional information to suggest that binge drinking patterns may be harmful for adolescent males, in terms of their brain function whilst completing executive function abilities. The need for further research exploring the effects of alcohol on executive functions in adolescent males is also highlighted. The present study also raises important questions about the criteria for binge drinking, and the need for an established consensus about what constitutes as binge drinking. This would be particularly helpful in order to make direct comparisons and conclusions between various studies that explore the psychological, behavioural and electrophysiological effects of heavy alcohol use.

Conclusion

The aim of the present study was to investigate if binge drinking is associated with deficits in behavioural and psychophysiological measures of interference suppression and response inhibition. The results of the present study indicated that despite no group differences in terms of accuracy and reaction time on the flanker/go-nogo task, low level drinkers displayed greater N2 amplitude at frontal than at central electrode sites, whereas binge drinkers did not display this difference. These results indicated that binge drinkers used more widespread recruitment of neural resources to inhibit their responses and filter out irrelevant stimuli on the flanker/go-nogo task than low level drinkers. Furthermore, these results suggested that binge drinkers recruit additional neural resources when using executive functions, however with no increase in task performance on the flanker/go-nogo task in comparison to low level drinkers. Despite some limitations, the present study raises some important questions about the electrophysiological effects of binge drinking in adolescent males. It is suggested that early intervention may be important for adolescent males in order to attempt to reduce binge drinking patterns during adolescence. Early intervention in the form of education to adolescent males and their parents of the risks associated with binge drinking patterns, and information to parents about how to monitor and attempt to reduce the amount of alcohol their adolescent child is drinking, would be ideal. Future research is also needed to clarify an appropriate and universally accepted definition of binge drinking, as well as to explore how binge drinking impacts on other executive functions such as planning and decision making in adolescence.

References

- Ahmadi, A., Pearlson, G. D., Meda, S. A., Dager, A., Potenza, M. N., Rosen, R., Austad, C., Raskin, S. A., Fallahi, C. R., Tennen, H., Wood, R. M., & Stevens, M. C. (2013). Influence of alcohol use on neural response to Go/No-Go task in college drinkers. *Neuropsychopharmacology*, 38(11), 2197-2208. doi: 10.1038/npp.2013.119
- Aiken, A., Wadolowski, M., Bruno, R., Najman, J., Kypri, K., Slade, T., Hutchinson D., McBride, N., & Mattick, R. P. (2015). Cohort profile: The Australian Parental Supply of Alcohol Longitudinal Study (APSALS). *International Journal of Epidemiology*. doi:10.1093/ije/dyv051
- Australian Institute of Health and Welfare, (2014). *National Drug Strategy Household Survey detailed report 2013* (pp. 1-150). Canberra: Australian Institute of Health and Welfare. Retrieved from <http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=60129549848>
- Beccaria, F., Petrilli, E., & Rolando, S. (2015). Binge drinking vs. drunkenness. The questionable threshold of excess for young Italians. *Journal of Youth Studies*, 18(7), 823-838. doi: 10.1080/13676261.2014.992321
- Bryan, J., & Luszcz, M. A. (2000). Measurement of Executive Function: Considerations for Detecting Adult Age Differences. *Journal of Clinical & Experimental Neuropsychology*, 22(1), 40-55. doi: 10.1076/1380-3395(200002)22:1;1-8;FT040
- Brydges, C. R., Clunies-Ross, K., Clohessy, M., Li Lo, Z., Nguyen, A., Rousset, C., Whitelaw, P., Jing Yeap Y. & Fox, A. (2012). Dissociable components of cognitive control: an event-related potential (ERP) study of response

inhibition and interference suppression. *Plos One*, 7(3), 34482. doi:
10.1371/journal.pone.0034482

Byrnes, J. P., Miller, D. C., & Schafer, W. D. (1999). Gender differences in risk taking: A meta-analysis. *Psychological Bulletin*, 125(3), 367-383. doi:
10.1037/0033-2909.125.3.367.

Cardenas, J. C., Dreber, A., von Essen, E., & Ranehill, E. (2012). Gender differences in competitiveness and risk taking: Comparing children in Colombia and Sweden. *Journal of Economic Behavior & Organization*, 83(1), 11-23. doi:
10.1016/j.jebo.2011.06.008

Casey, B., & Jones, R. (2010). Neurobiology of the adolescent brain and behavior: implications for substance use disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, 49(12), 1189--1201.
doi:10.1016/j.jaac.2010.08.017.

Clark, L., & Manes, F. (2004). Social and Emotional Decision-making Following Frontal Lobe Injury. *Neurocase (Psychology Press)*, 10(5), 398-403. doi:
10.1080/13554790490882799

Crego, A., Cadaveira, F., Parada, M., Corral, M., Caamaño-Isorna, F., & Rodríguez Holguín, S. (2012). Increased amplitude of P3 event-related potential in young binge drinkers. *Alcohol*, 46(5), 415-425. doi:
10.1016/j.alcohol.2011.10.002

Crego, A., Rodriguez Holguin, S., Parada, M., Mota, N., Corral, M., & Cadaveira, F. (2009). Binge drinking affects attentional and visual working memory processing in young university students. *Alcoholism: Clinical and Experimental Research*, 33(11), 1870 – 1879. doi: 10.1111/j.1530-0277.2009.01025.x

- Dayan, J., Bernard, A., Olliac, B., Mailhes, A. S., & Kerrmarec, S. (2010). Adolescent brain development, risk-taking and vulnerability to addiction. *Journal of Physiology- Paris*, 104, 279- 286.
doi:10.1016/j.jphysparis.2010.08.007
- De Bellis, M. D., Narasimhan, A., Thatcher, D. L., Keshavan, M. S., Soloff, P., & Clark, D.B. (2005). Prefrontal cortex, thalamus, and cerebellar volumes in adolescent and young adults with adolescent-onset alcohol use disorders and comorbid mental disorders. *Alcoholism: Clinical and Experimental Research*, 29(9), 159-600. doi: 10.1097/01.alc.0000179368.87886.76
- Doallo, S., Cadaveira, F., Corral, M., Mota, N., López-Caneda, E., & Holguín, S. R. (2014). Larger Mid-Dorsolateral Prefrontal Gray Matter Volume in Young Binge Drinkers Revealed by Voxel-Based Morphometry. *PLoS ONE*, 9(5), 1-8. doi: 10.1371/journal.pone.0096380
- Donkers, F. C. L., & van Boxtel, G. J. M. (2004). The N2 in go/no-go tasks reflects conflict monitoring not response inhibition. *Brain and Cognition*, 56(2), 165-176. doi: 10.1016/j.bandc.2004.04.005
- Easdon, C., Izenberg, A., Armilio, M. L., Yu, H., & Alain, C. (2005). Alcohol consumption impairs stimulus- and error-related processing during a Go/No-Go Task. *Cognitive Brain Research*, 25(3), 873-883. doi: 10.1016/j.cogbrainres.2005.09.009
- Eriksen, B. A., & Eriksen, C.W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics*, 16(1), 143-149. doi:10.3758/BF03203267
- Fellows, L. K. (2011). Orbitofrontal contributions to value-based decision making: evidence from humans with frontal lobe damage. *Annals of the New York*

Academy of Sciences, 1239(1), 51-58. doi: 10.1111/j.1749-6632.2011.06229.x

Fernie, G., Cole, J. C., Goudie, A. J., & Field, M. (2010). Risk-taking but not response inhibition or delay discounting predict alcohol consumption in social drinkers. *Drug and Alcohol Dependence*, 112(1-2), 54-61. doi: 10.1016/j.drugalcdep.2010.05.011

Ganguli, M., Vander Bilt, J., Saxton, J. A., Shen, C., & Dodge, H. H. (2005). Alcohol consumption and cognitive function in late life. *Neurobiology*, 65(8), 1210- 1217. doi: 10.1212/01.wnl.0000180520.35181.24

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A. Paus, T., Evans, A. C. & Rapoport, J. L. (1999). Brain development during childhood and adolescence: a longitudinal MRI study. *Nature Neuroscience*, 2(10), 861-863. doi:10.1038/13158

Hunt, W. A. (1993). Are binge drinkers more at risk of developing brain damage? *Alcohol*, 10(6), 559-561. doi:10.1016/0741-8329(93)90083-Z

Hutter, R. R. C., Lawton, R., Pals, E., O'Connor, D. B., & McEachan, R. R. C. (2015). Tackling student binge drinking: Pairing incongruent messages and measures reduces alcohol consumption. *British Journal of Health Psychology*, 20(3), 498-513. doi:10.1111/bjhp.12111

Isralowitz, R., Reznik, A., & Belhassen, Y. (2012). Binge drinking among Israeli hospitality workers: Gender and country of origin differences. *Drugs: Education, Prevention & Policy*, 19(2), 181-183. doi: 10.3109/09687637.2011.649498

- James, I. A., Reichelt, F. K., Carlsson, P., & McAnaney, A. (2008). Cognitive Behavior Therapy and Executive Functioning in Depression. *Journal of Cognitive Psychotherapy*, 22(3), 210-218. doi: 10.1891/0889-8391.22.3.210
- Jasper, H. H. (1958). The ten-twenty electrode system of the International Federation. *Electroencephalography and Clinical Neuropsychology*, 10, 370-375.
- Johansson, B., Berglund, P., & Rönnbäck, L. (2009). Mental fatigue and impaired information processing after mild and moderate traumatic brain injury. *Brain Injury*, 23(13-14), 1027-1040. doi: 10.3109/02699050903421099
- Kok, A., Ramautar, J., De Ruiter, M., B., & Ridderinkhof, K. (2004). ERP components associated with successful and unsuccessful stopping in a stop-signal task. *Psychophysiology*, 41(1), 9--20. doi: 10.1046/j.1469-8986.2003.00127.x
- Luciana, M., Collins, P. F., Muetzel, R. L., & Lim, K. O. (2013). Effects of alcohol use initiation on brain structure in typically developing adolescents. *The American Journal of Drug and Alcohol Abuse*, 39(6), 345-355. doi: 10.3109/00952990.2013.837057
- Luijten, M., Marise, W. J., Veltman, D. J., Hester, R., de Haan, L., Franken, I.H.A. (2014). Systematic review of ERP and fMRI studies investigating inhibitory control and error processing in people with substance dependence and behavioural addictions. *Journal of Psychiatry and Neuroscience*, 39(3), 149-169. doi: 10.1503/jpn.130052
- Mashhoon, Y., Czerkawski, C., Crowley, D. J., Cohen-Gilbert, J. E., Sneider, J. T., & Silveri, M. M. (2014). Binge alcohol consumption in emerging adults: Anterior cingulate cortical 'thinness' is associated with alcohol use patterns.

Alcoholism: Clinical and Experimental Research, 38(7), 1955-1964. doi:
10.1111/acer.12475

Maurage, P., Joassin, F., Speth, A., Modave, J., Philippot, P., & Campanella, S.

(2012). Cerebral effects of binge drinking: Respective influences of global alcohol intake and consumption pattern. *Clinical Neurophysiology*, 123(5), 892-901. doi: 10.1016/j.clinph.2011.09.018

Maurage, P., Pesenti, M., Philippot, P., Joassin, F., & Campanella, S. (2009). Latent deleterious effects of binge drinking over a short period of time revealed only by electrophysiological measures. *Journal of Psychiatry & Neuroscience*, 34(2), 111-118. doi:10.1016/s1053-8119(09)70040-0

Medina, K.L., McQueeny, T., Nagel, B.J., Hanson, K.L., Schweinburg, A.D., & Tapert, S.F. (2008). Prefrontal cortex volumes in adolescents with alcohol use disorders: unique gender effect. *Alcoholism: Clinical Experimental Research*, 32(3), 386-394. doi: 10.1111/j.1530-0277.2007.00602.x

Miller, J. W., Naimi, T. S, Brewer, R. D., & Jones, S. E. (2007). Binge Drinking and Associated Health Risk Behaviors Among High School Students. *Pediatrics*, 119(1), 76-85. doi: 10.1542/peds.2006-1517

Monti, P. M., Miranda, R., Jr., Nixon, K., Sher, K. J., Swartzwelder, H. S., Tapert, S. F., White, A. & Crews, F. T. (2005). Adolescence: Booze, Brains, and Behavior. *Alcoholism: Clinical and Experimental Research*, 29(2), 207-220. doi:10.1097/01.alc.0000153551.11000.f3

Mullan, B., Wong, C., Allom, V., & Pack, S. L. (2011). The role of executive function in bridging the intention-behaviour gap for binge-drinking in university students. *Addictive Behaviors*, 36(10), 1023-1026. doi:http://dx.doi.org/10.1016/j.addbeh.2011.05.012

- National Health and Medical Research Council (NHMRC), Australian guidelines to reduce health risks from drinking alcohol. 2009: Canberra.
- Parada, M., Corral, M., Mota, N., Crego, A., Rodríguez Holguín, S., & Cadaveira, F. (2012). Executive functioning and alcohol binge drinking in university students. *Addictive Behaviors*, 37(2), 167-172. doi: 10.1016/j.addbeh.2011.09.015
- Patrick, C. J., Bernat, E. M., Malone, S. M., Iacono, W. G., Krueger, R. F., & McGue, M. (2006). P300 amplitude as an indicator of externalizing in adolescent males. *Psychophysiology*, 43(1), 84-92. doi: 10.1111/j.1469-8986.2006.00376.x
- Paulus, M. P., Tapert, S. F., Pulido, C., & Schuckit, M. A. (2006). Alcohol attenuates load-related activation during a working memory task: Relation to level of response to alcohol. *Alcoholism: Clinical and Experimental Research*, 30(8), 1363-1371. doi: 10.1111/j.1530-0277.2006.00164.x
- Peraza, J., Cservenka, A., Herting, M. M., & Nagel, B. J. (2015). Atypical parietal lobe activity to subliminal faces in youth with a family history of alcoholism. *The American Journal of Drug and Alcohol Abuse*, 41(2), 139-145. doi: 10.3109/00952990.2014.953251.
- Petit, G., Maurage, P., Kornreich, C., Verbanck, P., & Campanella, S. (2013). Binge drinking in adolescents: a review of neurophysiological and neuroimaging research. *Alcohol and Alcoholism*, 49(2), 198-206. doi: 10.1093/alcalc/agt172
- Pfefferbaum, A., Adalsteinsson, E., & Sullivan, E. V. (2006). Dysmorphology and microstructural degradation of the corpus callosum: Interaction of age and alcoholism. *Neurobiology of Aging*, 27(7), 994-1009. doi:10.1016/j.neurobiolaging.2005.05.007

- Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology*, 118, 2128-2148. doi:10.1016/j.clinph.2007.04.019
- Polich, J., & Herbst, K. L. (2000). P300 as a clinical assay: Rationale, evaluation, and findings. *International Journal of Psychophysiology* 38, 3–19. doi: 10.1016/S0167-8760(00)00127-6
- Pujol, J., Vendrell, P., Junque, C., Mari-Vilalta, J., & Capdevila, A. (1993). When does human brain development end? Evidence of corpus callosum growth up to adulthood. *Annals Of Neurology*, 34(1), 71--75. doi: 10.1002/ana.410340113
- Reilly, D., van Beurden, E., Mitchell, E., Dight, R., Scott, C., & Beard, J. (1998). Alcohol education in licensed premises using brief intervention strategies. *Addiction*, 93(3), 385-398. doi: 10.1046/j.1360-0443.1998.9333857.x
- Renner, P., O'Dea, B., Sheehan, J., & Tebbutt, J. (2015). Days out of role in university students: The association of demographics, binge drinking, and psychological risk factors. *Australian Journal of Psychology*, 67(3), 157-165. doi:10.1111/ajpy.12077
- Risher, M., Fleming, R. L., Risher, W. C., Miller, K. M., Klein, R., & Wills, T., Acheson, S. K., Moore, S. D., Wilson, W. A., Eroglu, C., & Swartzelder, H. S. (2015). Adolescent Intermittent Alcohol Exposure: Persistence of Structural and Functional Hippocampal Abnormalities into Adulthood. *Alcoholism: Clinical and Experimental Research*, 39(6), 989-997. doi: 10.1111/acer.12725
- Roche, A. M., Bywood, P., Borlagdan, J., Lunnay, B., Freeman, T., Lawton, L., Tovell, A., & Nicholas, R. (2007). Young people and alcohol: The role of

cultural influences. National Centre for Education and Training on Addiction:
Adelaide

- Rubio, G., Rodriguez- Jimenez, R., Martinez, I., Aila, C., Ferre, F., Jimenez-Arriero, A., Ponce, G., & Palomo, T. (2008). The role of behavioral impulsivity in the development of alcohol dependence: A 4-year follow- up study. *Alcoholism: Clinical and Experimental Research*, 32, 1681- 1687. doi: 10.1111/j.1530-0277.2008.00746.x
- Russo, A. C. (2003). Executive Functioning and the Frontal Lobes: The Measure of Man? *Journal of Psycholinguistic Research*, 32(5), 567-571.
doi:10.1023/A:1025454501181
- Scaife J. C., & Duka, T. (2009). Behavioural measures of frontal lobe function in a population of young drinkers with binge drinking pattern. *Pharmacology Biochemistry and Behavior*, 93(3), 354-362. doi: 10.1016/j.pbb.2009.05.015
- Sneider, J. T., & Silveri, M. M. (2015). Neurobiology of teen brain development and the digital age. In M. Grabowski & M. Grabowski (Eds.), *Neuroscience and media: New understandings and representations*. (pp. 29-45). New York, NY, US: Routledge/Taylor & Francis Group.
- Sokhadze, E., Stewart, C., Hollifield, M., & Tasman, A. (2008). Event-related potential study of executive dysfunctions in a speeded reaction task in cocaine addiction. *Journal of Neurotherapy*, 12(4), 185-204. doi: 10.1080/10874200802502144
- Sumich, A., Kumari, V., Dodd, P., Ettinger, U., Hughes, C., Zachariah, E., & Sharma, T. (2008). N100 and P300 amplitude to Go and No-Go variants of the auditory oddball in siblings discordant for schizophrenia. *Schizophrenia Research*, 98(1-3), 265-277. doi: 10.1016/j.schres.2007.09.018

- Sur, S., & Sinha, V. K. (2009). Event-related potential: An overview. *Industrial Psychiatry Journal*, 18(1), 70-73. doi: 10.4103/0972-6748.57865
- Thayanukulvat, C., & Harding, T. (2015). Binge drinking and cognitive impairment in young people. *British journal of nursing*, 24(7), 401-407.
doi:10.12968/bjon.2015.24.7.401
- Townshend, J. M., & Duka, T. (2005). Binge Drinking, Cognitive Performance and Mood in a Population of Young Social Drinkers. *Alcoholism: Clinical and Experimental Research*, 29(3), 317-325. doi:
10.1097/01.alc.0000156453.05028.f5
- Townshend, J. M., Kambouropoulos, N., Griffin, A., Hunt, F. J., & Milani, R. M. (2014). Binge drinking, reflection impulsivity, and unplanned sexual behavior: Impaired decision-making in young social drinkers. *Alcoholism: Clinical and Experimental Research*, 38(4), 1143-1150.
doi:10.1111/acer.12333
- Wechsler, D. (2001). *Wechsler Test of Adult Reading: WTAR*. Psychological Corporation.
- Wilcox, C. E., Dekonenko, C. J., Mayer, A. R., Bogenschutz, M. P., & Turner, J. A. (2014). Cognitive control in alcohol use disorder: Deficits and clinical relevance. *Reviews in the Neurosciences*, 25(1), 1-24.
doi:10.1016/j.alcohol.2014.07.020

Table of Appendices

Appendix A: Email to Participants.....	54
Appendix B: Screening Questionnaire.....	57
Appendix C: Human Research Ethics Committee (HREC) Approval from the University of Tasmania.....	65
Appendix D: Information Sheet	68
Appendix E: Informed Consent.....	72
Appendix F: Data Analyses.....	77

Appendix A

Email to Participants

Hello,

Thank you for your participation in the 'Drinking and Teens' study – we really appreciate your ongoing contribution.

In the last round of questionnaires, you said that you would be interested in hearing more about future opportunities to participate in related research studies. So, we are writing to tell you about a new study that relates to the 'drinking and teens' study, which is designed to understand the effects that alcohol might have on cognition (the way that you solve problems and learn new information) in teens. As part of this study we're interested in assessing people who have had no alcohol, small amounts of alcohol and people that have had larger amounts of alcohol. We are wondering if your son would be interested in participating in the following study.

This study will be conducted at the Cognitive Neuroscience Laboratory in the Psychology Research Centre, at the Hobart Campus of the University of Tasmania.

Taking part in the study would take a couple of hours of your sons time. It would involve completing a short questionnaire about his health and any alcohol use since he completed the last survey for the 'drinking and teens' study to make sure that he is eligible. After that, he would be invited to come to the university for around a two hour session, where he would be asked to complete some simple tasks on a computer while his brain activity is measured.



This would involve wearing a cap like this, which is covered in electrodes to record the activity of your brain while you are completing the tasks. From this, we can tell what parts of the brain are being used to complete different parts of each task, and how active they are. The electrodes sit on top of the skin so there's nothing invasive about the recording.

If your son would like to take part in the study, please direct him to the following link: <https://surveys.psychol.utas.edu.au/index.php/789216/lang-en> to complete a short (approximately 5-10 minutes) screening questionnaire and we will then contact him and invite him to come to the university. If you would like more information about the study please send us a reply by email or call us on the phone number below.

We have attached a copy of the information sheet about the study to this email as well. And you will be reimbursed \$40 at the end of the study for your time and participation.

Thank you for your time in considering this study, we hope that you will be interested in taking part.

Kind regards,

Michelle Dwyer

University of Tasmania

Phone:

Email: Michelle.Dwyer@utas.edu.au

Appendix B
Screening Questionnaire

CANDY ERP Screening Questionnaire

Brief screening questionnaire

Hello, thank you for choosing to participate in the ‘Drinking and Teens’ study – we really appreciate your ongoing contribution.

This study is designed to understand the effects that alcohol might have on cognition (the way that you solve problems and learn new information). As part of this study we’re interested in assessing people who have had no alcohol, small amounts of alcohol and people that have had larger amounts of alcohol.

This questionnaire is a very brief (approximately 5-10 minutes) questionnaire asking some questions in relation to your general health, as well as your amount of alcohol consumption in the last 12 months, and your availabilities of when you would like to come in to the university to participate in the study.

Once you have completed this brief questionnaire, if you are eligible for the study, we will contact you to organise a time that suits you to come into the Psychology Research Centre, at the Hobart Campus of the University of Tasmania to complete the experiment part of the study, where you will be asked to complete some simple tasks on a computer while your brain activity is measured using an electrode cap.

All data will be stored securely and password-protected and will be kept confidential. All data will be kept up to a minimum period of 5 years from the date of research publication and will be destroyed/removed after 5 years.

If you have any questions or concerns about this study please contact Michelle Dwyer (Michelle.Dwyer@utas.edu.au) or Raimondo Bruno (Raimondo.Bruno@utas.edu.au)

Thank you for your time in completing this brief questionnaire, as well as your interest in engaging in the next part of the study.

Contact Details

Full Name:

Please indicate your preferred contact number for us to contact you on:

Which day would you prefer us to contact you on? (You can specify the times you would like to be called if you like)

Please choose all that apply

- ☐ Monday
- ☐ Tuesday
- ☐ Wednesday

- ☐ Thursday
- ☐ Friday
- ☐ Saturday
- ☐ Sunday
- ☐ Any day

Demographic Information

Date of Birth:

Are you right or left handed?

- ☐ Right Handed
- ☐ Left Handed

Are you currently taking any prescription medication?

- ☐ Yes
- ☐ No

Please specify (e.g. the type of medication and if you take it every day)

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '6 [3]' (Are you currently taking any prescription medication?)

(if you don't wish to specify, that's OK, please just put a dot in the text box)

Have you ever suffered from an epileptic seizure?

- ☐ Yes
- ☐ No

Please specify (e.g. how long since the last time and how frequently this happens)

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '8 [4]' (Have you ever suffered from an epileptic seizure?)

(if you don't wish to specify, that's OK, please just put a dot in the text box)

Do you often experience giddiness or fainting?

- ☐ Yes
- ☐ No

Please specify (e.g. how long since the last time and how frequently this happens)

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '10 [5]' (Do you often experience giddiness or fainting?)

(if you don't wish to specify, that's OK, please just put a dot in the text box)

Have you ever suffered from a serious head injury, concussion or periods of unconsciousness? *

- ☐ Yes
- ☐ No

Please specify (e.g. the type of event and how long since the last time)

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '12 [6]' (Have you ever suffered from a serious head injury, concussion or periods of unconsciousness?)

(if you don't wish to specify, that's OK, please just put a dot in the text box)

Do you have any other serious physical condition?

- ☐ Yes
- ☐ No

Please specify the type of condition:

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '14 [7]' (Do you have any other serious physical condition?)

(if you don't wish to specify, that's OK, please just put a dot in the text box)

Have you ever suffered from anxiety or depression or any other mental health condition?

- ☐ Yes

- ☐ No

Please specify (e.g. if you are taking any medications or if you are seeing anyone for help)

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '16 [8]' (Have you ever suffered from anxiety or depression or any other mental health condition?)

(if you don't wish to specify, that's OK, please just put a dot in the text box)

Do you have any hearing problems?

- ☐ Yes
- ☐ No

Please specify the hearing problem and if it is corrected (for example by hearing aids)

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '18 [9]' (Do you have any hearing problems?)

(if you don't wish to specify, that's OK, please just put a dot in the text box)

Do you have any vision problems?

- ☐ Yes
- ☐ No

Please specify the vision problem and if it is corrected by glasses/contact lenses:

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '20 [10]' (Do you have any vision problems?)

(if you don't wish to specify, that's OK, please just put a dot in the text box)

Do you have very sensitive skin or are you allergic to any skin products?

- ☐ Yes
- ☐ No

Please specify the types of things that cause you skin problems

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '22 [11]' (Do you have very sensitive skin or are you allergic to any skin products?)

Do you have a current sleep disorder or serious sleeping difficulties?

- ☐ Yes
- ☐ No

Please specify (e.g. how much sleep a night you get on average over the last few days)

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '24 [12]' (Do you have a current sleep disorder or serious sleeping difficulties?)

How often do you smoke tobacco?

- ☐ Never
- ☐ Monthly
- ☐ Fortnightly
- ☐ Weekly
- ☐ Daily or almost daily

Are you from a non-English speaking background?

- ☐ Yes
- ☐ No

Alcohol Use

Have you had an alcoholic beverage in the last 12 months?

- ☐ Yes
- ☐ No

Within the last 12 months, how often have you had an alcohol drink of any kind?

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '28 [15]' (Have you had an alcoholic beverage in the last 12 months?)

- ☐ 5 or more days a week
- ☐ 1-4 days a week
- ☐ 1-3 days a month
- ☐ less often but at least once

In the last 12 months how often have you had more than 4 standard drinks in a day? (see Standard Drinks Guide)

STANDARD DRINKS GUIDE			
 1 285ml Middy Full Strength Beer	 0.7 285ml Middy Mid Strength Beer	 0.5 285ml Middy Light Beer	 1.5 170ml Sparkling Wine
 1.5 375ml Full Strength Beer	 1 375ml Mid Strength Beer	 0.8 375ml Light Beer	 1.5 375ml pre-mix spirits
 1.5 340ml Alcoholic Soda	 1 30ml Spirit Nip	 22 700ml Bottle of Spirits	 0.9 60ml Port/Sherry glass
 1 100ml Wine	 1.8 170ml Sparkling Wine/Champ	 7 750ml Bottle of Wine	 38 4 Litre Cask Wine

Only answer this question if the following conditions are met:

Answer was 'Yes' at question '28 [15]' (Have you had an alcoholic beverage in the last 12 months?)

- ☐ At Least Once
- ☐ Never

In the last 12 months, how often have you had more than 4 standard drinks in a day?

Only answer this question if the following conditions are met:

Answer was 'At Least Once' at question '30 [17]' (In the last 12 months how often have you had more than 4 standard drinks in a day? (see Standard Drinks Guide))

- ☐ 5 or more days a week
- ☐ 1-4 days a week
- ☐ 1-3 days a month
- ☐ less often but at least once

In the last 12 months, how often have you used any of the following substances?
(Please tick)

Substance	Never	Once	2-5 Times	6 or more times
Cigarettes/ Tobacco				
Cannabis / Marijuana				
Ecstasy				
Speed / Amphetamines				
Other Substances: _____				

Appendix C

Human Research Ethics Committee (HREC) Approval from the
University of Tasmania

Social Science Ethics Officer
Private Bag 01 Hobart
Tasmania 7001 Australia
Tel: (03) 6226 2763
Fax: (03) 6226 7148
Katherine.Shaw@utas.edu.au



HUMAN RESEARCH ETHICS COMMITTEE (TASMANIA) NETWORK

20 September 2013

Dr Raimondo Bruno
School of Psychology
Private Bag 30

Student Researcher: Emilia Gencic

Sent via email

Dear Dr Bruno

Re: FULL ETHICS APPLICATION APPROVAL
Ethics Ref: H0013436 - Consumption of Alcohol and Executive Function in Youth

We are pleased to advise that the Tasmania Social Sciences Human Research Ethics Committee approved the above project on 18 September 2013.

This approval constitutes ethical clearance by the Tasmania Social Sciences Human Research Ethics Committee. The decision and authority to commence the associated research may be dependent on factors beyond the remit of the ethics review process. For example, your research may need ethics clearance from other organisations or review by your research governance coordinator or Head of Department. It is your responsibility to find out if the approval of other bodies or authorities is required. It is recommended that the proposed research should not commence until you have satisfied these requirements.

Please note that this approval is for four years and is conditional upon receipt of an annual Progress Report. Ethics approval for this project will lapse if a Progress Report is not submitted.

The following conditions apply to this approval. Failure to abide by these conditions may result in suspension or discontinuation of approval.

1. It is the responsibility of the Chief Investigator to ensure that all investigators are aware of the terms of approval, to ensure the project is conducted as approved by the Ethics Committee, and to notify the Committee if any investigators are added to, or cease involvement with, the project.

A PARTNERSHIP PROGRAM IN CONJUNCTION WITH THE DEPARTMENT OF HEALTH AND HUMAN SERVICES

2. Complaints: If any complaints are received or ethical issues arise during the course of the project, investigators should advise the Executive Officer of the Ethics Committee on 03 6226 7479 or human.ethics@utas.edu.au.
3. Incidents or adverse effects: Investigators should notify the Ethics Committee immediately of any serious or unexpected adverse effects on participants or unforeseen events affecting the ethical acceptability of the project.
4. Amendments to Project: Modifications to the project must not proceed until approval is obtained from the Ethics Committee. Please submit an Amendment Form (available on our website) to notify the Ethics Committee of the proposed modifications.
5. Annual Report: Continued approval for this project is dependent on the submission of a Progress Report by the anniversary date of your approval. You will be sent a courtesy reminder closer to this date. Failure to submit a Progress Report will mean that ethics approval for this project will lapse.
6. Final Report: A Final Report and a copy of any published material arising from the project, either in full or abstract, must be provided at the end of the project.

Yours sincerely

Katherine Shaw
Ethics Officer
Tasmania Social Sciences HREC

Appendix D
Information Sheet



School of Psychology
University of Tasmania

Information Sheet

How does alcohol impact on executive functioning in young people?

What is this study about?

This study is trying to understand whether low and high levels of alcohol consumption during adolescence have any impacts on a particular aspect of cognition, called executive function. This is important when you're working out how to solve problems, use strategies to learn new information, and to control your concentration. This study will use special recording processes to measure brain activity while people complete different tasks of 'executive function'.

Who is involved?

This study is being conducted by the 'Drinking and Teens' team, as well as, locally, Michelle Dwyer, a Psychology Clinical Masters student, under the supervision of Dr Raimondo Bruno.

Who can take part?

We've contacted you because you have been taking part in the 'Drinking and Teens' study over the past couple of years and told us that you were interested in hearing more about other studies we're doing that are related to this project. We're interested in talking with people that have had no alcohol, people who have had only low levels of alcohol, as well as people that have had larger amounts from time to time. While this includes everybody who is taking part in the 'Drinking and Teens' study, to make the results of the study clear, we are looking specifically for males aged approximately 15 -17.

What would I have to do?

If you take part, we would invite you to complete an online survey to answer a few questions about your health and to update us on any alcohol you've had since you last completed the 'drinking and teens' questionnaire. These questions allow us to check to make sure that you don't have any health issues which might impact on the assessments.

From there, you'll be invited to come to the Psychology Research Centre at the Sandy Bay Campus for a two hour session. This will include things like reading aloud a list of unusual words; and learning long lists of words. After this, we'll fit an electrode cap on your head which can measure your brain activity (the electrodes sit on top of your scalp to do this, there's nothing that sticks in to you at all). Once this is set up correctly, you would be asked to complete a few simple computer tasks like responding as quickly as possible when an arrow appears on a computer screen, while there's distracting information on the monitor at the same time. While you do the tasks your brain activity will be recorded which will let us know what parts of the brain are active as you complete the tasks, and how active they are.

What are the benefits of taking part?

Your participation will help us understand whether alcohol has particular effects on these 'executive' tasks. This will help in the education of people about the effects of drinking during teenage years, and help to guide public policy and drinking guidelines.

Are there any negatives that I need to know about?

The electrode cap can feel a little uncomfortable, but it is not painful in any way. However, if you have sensitive skin, please let us know as we need to use some gel to clean the skin where the electrodes sit and to get a clear recording of your brain waves. This gel may cause irritation if you have sensitive skin. There are no anticipated risks of the study, but in the unlikely event that you do experience any negative side effects, please tell us immediately and any necessary assistance will be provided.

Do I have to take part? What if I start and then change my mind?

Participation in this study is completely voluntary. By signing the attached consent form, you are indicating that you are aware of the nature of the study and wish to participate. While we would be pleased to have you participate, we completely respect your right to decline. There will be no consequences to you if you decide not to participate. Likewise, if you start and then decide that you'd like to discontinue at any stage, that's completely fine and you wouldn't need to explain why.

I'd like to help but it's a bit expensive to get in to the university

To cover the costs associated with your time and transport into the university, all participants will be provided with \$40 at the end of the session.

Is the information confidential?

All information collected will be kept confidential. Each participant will be assigned a treatment code and individual participant data will be identifiable only by that code. All of the data will be stored on password protected secure computers or in a locked cabinet in the School of Psychology for a minimum of five years after

the publication of any academic journal articles, at which point all questionnaires will be destroyed using a paper shredder and electronic data will be deleted. In any publication that is written, no individual would be identified (only the average performance of groups of people are used).

Who do I talk to if I want to know more?

If you would like to discuss any aspect of this study please contact Michelle Dwyer on email Michelle.Dwyer@utas.edu.au. Alternatively, you can contact Dr Raimondo Bruno on (03) 6226 2240 or email Raimondo.Bruno@utas.edu.au. A summary of the results will be available on the Research webpage of the School of Psychology, University of Tasmania (www.utas.edu.au/psychology/). Results of the study can also be provided by Michelle Dwyer (Michelle.Dwyer@utas.edu.au)

Has this all been checked to make sure that the research is ethical?

This study has been approved by the Tasmanian Social Science Human Research Ethics Committee. If you have concerns or complaints about the conduct of this study should contact the Executive Officer of the HREC (Tasmania) Network on (03) 6226 7479 or email human.ethics@utas.edu.au. The Executive Officer is the person nominated to receive complaints from research participants. You will need to quote H0013436.

Thank you for taking the time to consider this study.

If you wish to take part in it, please sign the attached consent form.

This information sheet is for you to keep.

Appendix E
Informed Consent



School of Psychology
University of Tasmania

Consent Form- Adolescent

Consumption of Alcohol and Executive Function in Youth

1. I have read and understood the 'Information Sheet' for this project.
2. The nature and possible effects of the study have been explained to me.
3. I understand that the study involves attending the Cognitive Neuroscience Laboratory for one two-hour session after completion of a medical history questionnaire, and record of recent alcohol consumption online to assess my eligibility.
4. I understand that I will be asked to abstain from alcohol for 24 hours prior to the testing session.
5. I understand that I will be fitted with an electrode cap, which will non-invasively record my brain activity from the scalp. I will be asked to complete a number of computerised laboratory behavioural performance tasks during which my behavioural responses and brain activity will be recorded.
6. I understand that, while there are no anticipated risks associated with this study, I should inform the experimenter immediately if any unexpected negative side-effects are experienced. I understand the experimenter will immediately cease the session and seek any necessary assistance.
7. I understand that all research data will be securely stored on the University of Tasmania premises for at least five years, and will then be securely destroyed when no longer required.
8. Any questions that I have asked have been answered to my satisfaction.
9. I agree that research data gathered from me for the study may be published provided that I cannot be identified as a participant.
10. I understand that the researchers will keep my identity confidential and that any information I supply to the researcher(s) will be used only for the purposes of the research. My data will only be identifiable by an individual numerical participant code.
11. I understand that researchers will access information I previously gave when participating in the "Drinking and Teens" study.

12. I agree to participate in this investigation and understand that I may withdraw at any time without any effect, and if I so wish, may request that any data I have supplied to date be withdrawn from the research.

Name of Participant: _____

Signature: _____

Date: _____

Statement by Investigator

- ☐ I have explained the project & the implications of participation in it to this volunteer and I believe that the consent is informed and that he/she understands the implications of participation

If the Investigator has not had an opportunity to talk to participants prior to them participating, the following must be ticked.

- ☐ The participant has received the Information Sheet where my details have been provided so participants have the opportunity to contact me prior to consenting to participate in this project.
- ☐ The participant has consented for their information from the "Drinking and Teens" study to be accessed.

Name of investigator: _____

Signature of investigator: _____

Date: _____



School of Psychology
University of Tasmania

Consent Form- Parent

Consumption of Alcohol and Executive Function in Youth

1. I have read and understood the 'Information Sheet' for this project.
2. The nature and possible effects of the study have been explained to me.
3. I understand that the study involves my son attending the Cognitive Neuroscience Laboratory for one two-hour session after completion of a confidential online medical history questionnaire, and record of recent alcohol consumption to assess his eligibility.
4. I understand that he will be asked to abstain from alcohol for 24 hours prior to the testing session.
5. I understand that he will be fitted with an electrode cap, which will non-invasively record his brain activity from the scalp. He will be asked to complete a number of computerised laboratory behavioural performance tasks during which his behavioural responses and brain activity will be recorded.
6. I understand that, while there are no anticipated risks associated with this study, he should inform the experimenter immediately if any unexpected negative side-effects are experienced. I understand the experimenter will immediately cease the session and seek the necessary assistance.
7. I understand that all research data will be securely stored on the University of Tasmania premises for at least five years, and will then be securely destroyed when no longer required.
8. Any questions that I have asked have been answered to my satisfaction.
9. I agree that research data gathered from him for the study may be published provided that he cannot be identified as a participant.
10. I understand that the researchers will keep his identity confidential and that any information he supplies to the researcher(s) will be used only for the purposes of the research. His data will only be identifiable by an individual numerical participant code.
11. I understand that researchers will access information he previously gave when participating in the "Drinking and Teens" study.

12. I agree for my son to participate in this investigation and understand that he or I may withdraw at any time without any effect, and if I so wish, may request that any data he have supplied to date be withdrawn from the research.

Name of Parent: _____

Signature: _____

Date: _____

Statement by Investigator

- ☐ I have explained the project & the implications of participation in it to this participant's parent and I believe that the consent is informed and that he/she understands the implications of participation

If the Investigator has not had an opportunity to talk to participants prior to them participating, the following must be ticked.

- ☐ The participant's parent has received the Information Sheet where my details have been provided so participants have the opportunity to contact me prior to consenting to participate in this project.

- ☐ The participant's parent has consented for their information from the "Drinking and Teens" study to be accessed.

Name of investigator: _____

Signature of investigator: _____

Date: _____

Appendix F

Data Analyses

See CD attached on the back cover for below contents:

All raw and demographic data – SPSS Data File

Behavioural Data

Behavioural data (Reaction Time and Accuracy) - SPSS Output

N2 Amplitude

N2 amplitude - SPSS Output

N2 amplitude Site X Trial interaction – SPSS Output

N2 amplitude Site X Group interaction – SPSS Output

N2 mean difference amplitude - SPSS Output

P3 Amplitude

P3 amplitude - SPSS Output

P3 amplitude Site x Trial Interaction - SPSS Output